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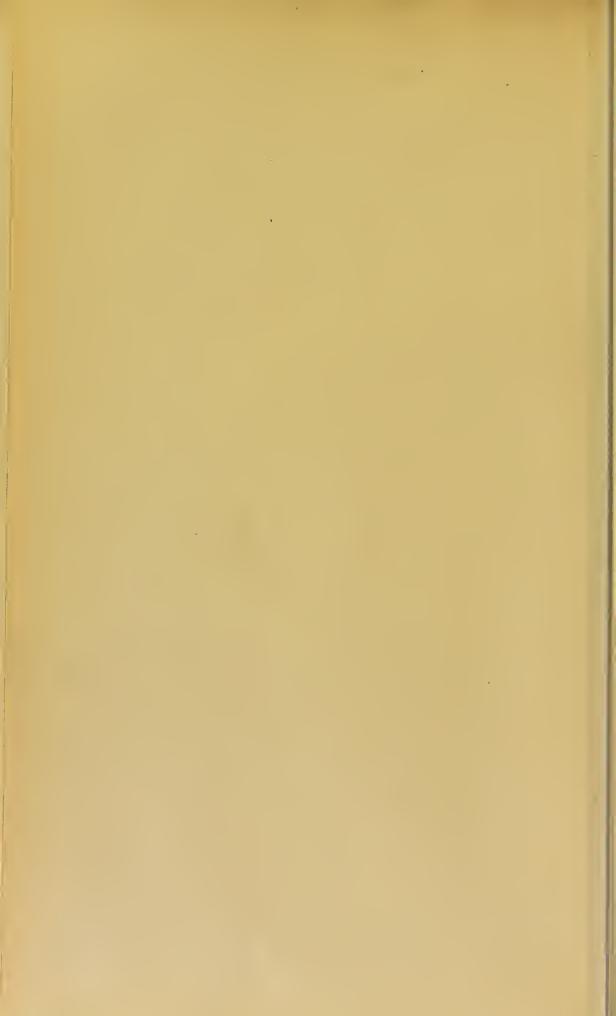




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ULCER

OF THE

STOMACH AND DUODENUM

BY THE SAME AUTHORS

CANCER OF THE STOMACH

(In preparation)

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ULCER

OF THE

STOMACH AND DUODENUM

AND ITS CONSEQUENCES

 $\mathbf{B}\mathbf{Y}$

SAMUEL | FENWICK, M.D., F.R.C.P.

CONSULTING PHYSICIAN TO THE LONDON HOSPITAL

AND

W. SOLTAU FENWICK, M.D. LOND., M.R.C.P.

SENIOR PHYSICIAN TO THE LONDON TEMPERANCE HOSPITAL PHYSICIAN TO THE EVELINA HOSPITAL FOR SICK CHILDREN



LONDON J. & A. CHURCHILL

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PREFACE

THE following work is divided into four parts, the first of which deals with the Pathology and Etiology of Gastric and Duodenal Ulcer, while the other three are devoted to a consideration of the clinical aspects of the disease and its sequelæ.

The conclusions respecting the morbid anatomy are principally derived from the post-mortem records of 1,015 cases of ulcer of the stomach and of 130 cases of ulcer of the duodenum. In order to avoid the errors that are apt to arise from the collection of isolated examples of the disease, the various cases have been taken exclusively from hospital records, and include 132 which were examined at the London Hospital and the London Temperance Hospital. One of the principal features of the analysis is the distinction which is made between the acute and chronic forms of the complaint as regards their origin, appearances, and modes of termination.

The second part contains a description of the symptoms, varieties, and treatment of Acute Ulceration of the Stomach and Duodenum, which is based upon the notes of 143 clinical cases, the majority of which were under the personal observation of the authors.

It is shown that the disease may either occur as an idiopathic affection, or ensue during the course of some other malady, the former being chiefly confined to young women, while the latter is not infrequently the unsuspected cause of death in certain infective complaints and in diseases of the heart and liver.

The third part deals with the symptomatology of the ordinary chronic ulcer, based upon an analysis of 308 clinical cases. Five varieties of the gastric disease are differentiated, and the difficulties involved in their prognosis, diagnosis, and treatment are discussed.

The final portion of the volume is devoted to the different sequelæ of ulceration, which have hitherto received less notice than they deserve. Special attention is directed to the effects of perigastric adhesions, to the deformities of the stomach that ensue from the cicatrisation of an ulcer, and to the various internal fistulæ.

A full account is also given of such subjects of practical interest as perigastric and periduodenal abscess, tetany and other nervous disorders, cancer, tuberculosis, pernicious anæmia, gastric catarrh, and hypersecretion. With regard to the latter complaint the belief is expressed that the excessive secretion of gastric juice is only a reflex result of the irritation produced by the presence of the ulcer, and reasons are given for believing that the so-called 'disease of Reichmann,' instead of being a primary functional disorder of the stomach, is merely a consequence of the retention of the gastric contents in certain forms of pyloric obstruction.

Although the work has chiefly been written from personal experience, every effort has been made to represent the views of others. At the end of the Third Part is appended a Bibliography of nearly six hundred references, while about one hundred and fifty relating to the sequelæ are contained in the last Part. The majority of these references have been verified and the subject matter perused, but unfortunately in many instances the original communications were not to be obtained. The labour of producing a correct bibliography was greatly

increased by the difficulty of identifying the names of many authors, owing to incorrect spelling and the ludicrous changes and alterations which they had suffered in the course of repeated transcription. In addition to this, many papers were found to be attributed to persons of whom no record could be discovered, while others were credited to individuals who never wrote them.

Most of the illustrations are from photographs made from museum preparations, and we are indebted to the Council of the Royal College of Surgeons and to the College Board of the London Hospital for kind permission to use the valuable material contained in their respective museums. We have also to express our thanks to Mr. L. Galsworthy, who kindly assisted us in the supervision of the proofs.

W. SOLTAU FENWICK.

8 Devonshire Street, London, W. February, 1900.



HISTORICAL INTRODUCTION

It is probable that simple ulceration of the stomach was known to the earliest writers on medicine, but it appears to have been generally confused by them with cancer and the effects of postmortem digestion.

According to Bonetus the first authentic case was recorded by Johann Bauhin about the middle of the sixteenth century, who described a perforation in the middle of the stomach of a young girl who had previously suffered from hæmatemesis. Towards the end of the century similar instances were published by Marcellus Donatus, Courtial, and Littré. About the same time the effects of the disease were also beginning to excite attention, for we find notices of cicatrices in the writings of Forestus and Schenk, while cases of hour-glass stricture and other deformities of the stomach from the same cause are recorded in those of Blasse, Mangold, Heister, Sandifort, Sömmering, and Morgagni. One of the first cases of gastric fistula appears in the memoir of Duverney (1704), and a communication between the stomach and the colon was observed by Haller in 1744. The presence of adhesions between the stomach and neighbouring organs was also commented upon by Mead, Murray, Santessen, and Westring.

In the year 1798 Baillie published some excellent drawings of simple ulcer of the stomach, and gave an accurate description of its principal features, which in 1824 was further amplified by Abercrombic, who also noted the occurrence of perforation of the duodenum. In the meantime Rausch, in his classification of ruptures of the stomach, had come to the conclusion

that a round ulcer with callous edges was one of the most frequent causes of the accident. It was left, however, to Cruveilhier to bring the various observations upon the disease into harmony, and to establish simple ulcer of the stomach as a pathological and clinical entity. In his great work, which made its appearance between 1829 and 1838, he not only described the general features of the complaint, with its modes of termination and sequelæ, but depicted its various symptoms and its appropriate method of treatment. It may truly be said that much of our present knowledge is still based upon these researches of Cruveilhier. Rokitanski's masterly survey of the disease appeared in 1839.

The method of collecting a large series of cases for the purposes of analysis appears to have originated with Jaksch and his immediate successors at Prague, and was extended with brilliant results by Brinton in his celebrated book on Ulcer of the Stomach, published in 1857. Since that time the most important contributions to our clinical knowledge are contained in the works of Samuel Fenwick, Wilson Fox, and Habershon in England; Lebert, Müller, Leube, and Ziemssen in Germany; Welch in America; and Trousseau and Bouveret in France.

As far back as 1800 Morin discussed the etiology of gastric ulcer, and attributed it to a local disturbance of the circulation of the stomach, but it was not until 1855 that Virchow propounded his theory of the embolic origin of the disease. During the last fifty years Panum, Müller, Cohnheim, Pavy, Ebstein, and a host of other experimental pathologists have done much to extend our knowledge of the pathogenesis of the complaint.

CONTENTS

	PAGE ix
PART I	
PATHOLOGY AND ETIOLOGY	
I. MORBID ANATOMY	1 28 78 97
PART II ACUTE ULCER OF THE STOMACH AND DUODENUM	
I. SYMPTOMATOLOGY	131 159 167
PART III	
CHRONIC ULCER OF THE STOMACH AND DUODENUM	
I. SYMPTOMATOLOGY	183 215 224 230 243

PART IV

THE SEQUELÆ OF CHRONIC ULCER

CHAPT	IR Control of the Con	PAGE
ı.	PERIGASTRIC ADHESIONS	. 269
II.	THE RESULTS OF CICATRISATION	. 277
III.	HYPERSECRETION AND GASTRIC CATARRH	. 299
IV.	TETANY AND OTHER NERVOUS DISORDERS	. 311
v.	PERIGASTRIC AND PERIDUODENAL ABSCESS	. 323
VI.	GASTRIC FISTULÆ	. 351
VII.	TUBERCULOSIS, CANCER, AND PERNICIOUS ANÆMIA	. 370
	INDEX	. 385

LIST OF ILLUSTRATIONS

FIG.		PAGE
1.	MULTIPLE ULCERS IN THE STOMACH	2
2.	ACUTE PERFORATING ULCER IN A YOUNG GIRL	
3.	A MINUTE ULGER NEAR THE CARDIAC ORIFICE	5
4.	AN IMMENSE CHRONIC ULCER OF THE STOMACH	6
5.	A PERFORATED ULCER WITH A SCAR ON THE OPPOSITE WALL .	9
6.	A PERFORATED ULCER WITH A SCAR THAT WAS DISCOVERED BY PHOTOGRAPHY	10
7.	A CRESCENTIC ULCER AT THE CARDIAC ORIFICE	11
8.	AN ULCER OF TRIANGULAR SHAPE	13
9.	A CHRONIC ULCER WITH A SCALLOPED EDGE	-4.4
10.	A HORSESHOE ULCER	14
11.	A SIMPLE CHRONIC ULCER OF THE STOMACH	21
12.	A CHRONIC ULCER WITH SLOPING EDGES	23
13.	A CHRONIC ULCER OF QUADRILATERAL FORM	25
14.	A PORTION OF THE PANCREAS PROJECTING THROUGH THE BASE OF	
	AN ULCER	30
15.	AN ACUTE ULCER NEAR THE CARDIAC ORIFICE, WITH TWO SCARS .	35
16.	A CICATRIX OF A CHRONIC ULCER	36
17.	CHRONIC ULCER EMBRACING THE CARDIAC ORIFICE	. 41
18.	The state of the s	
	PYLORUS	
19.	HOUR-GLASS STOMACH OF CONGENITAL ORIGIN	. 45
20.	The second of th	
0.4	MUCOUS MEMBRANE	
21.	A RECESSED ULCER OF THE DUODENUM WITH A LARGE POUCH	
22.	The Blomkett And Doorens	
23.		. 52
24.		
25.		
26.	ADSORDA	
	CHRONIC ULCER ERODING THE PANCREAS	. 67
28.	PERFORATION OF THE DIAPHRAGM BY AN ULCER	. 68

xiv LIST OF ILLUSTRATIONS

FIG.		PAGE
29.	PERFORATION OF THE COLON BY AN ULCER	
30.	ACUTE ULCERATION OF THE STOMACH IN PYÆMIA	102
31.	ULCERATION OF THE STOMACH AND DUODENUM AFTER BURNS .	110
32.	ENLARGEMENT OF THE SOLITARY GLANDS OF THE STOMACH	120
33.	SECTION OF A STOMACH AFFECTED WITH FOLLICULAR ULCERATION.	121
34.	SECTION OF A STOMACH SHOWING AN ENLARGED SOLITARY GLAND.	121
35.	GASTRIC ULCERATION IN ENTERIC FEVER	122
36.	INFLAMMATION OF THE SOLITARY GLANDS OF THE STOMACH WITH	
	ULCERATION	123
37.	TEMPERATURE CHART AFTER HÆMATEMESIS	137
38.	TEMPERATURE CHART IN GENERAL PERITONITIS	143
39.	TEMPERATURE CHART IN PERFORATION OF THE STOMACH WITH	
	SPONTANEOUS RECOVERY	145
40.	ACUTE ULCERS OF THE STOMACH IN PYÆMIA	147
41.	ACUTE ULCERS OF THE STOMACH IN ENTERIC FEVER	149
42.	AN ACUTE ULCER NEAR THE CARDIAC ORIFICE LEADING TO FATAL	
	HÆMORRHAGE	153
43.	VARICOSE VEINS IN THE STOMACH WITH ACUTE ULCERATION	154
44.	DIAGRAM OF THE SITE OF THE EPIGASTRIC PAIN IN CHRONIC ULCER	185
45.	DIAGRAM OF THE SITE OF THE DORSAL PAIN	186
46.	DIAGRAM OF THE ARTERIAL BLOOD-SUPPLY OF THE STOMACH .	196
47.	TEMPERATURE CHART IN A CASE OF REPEATED HÆMATEMESIS .	197
48.	TUMOUR IN THE STOMACH FORMED BY THE PANCREAS	210
49.	STENOSIS OF THE PYLORIC ORIFICE	278
50.	AN ULCER OF THE CARDIAC ORIFICE WHICH GAVE RISE TO DYSPHAGIA	284
51.	HOUR-GLASS STOMACH	292
52.	DIAGRAM OF A CASE OF PERIGASTRIC ABSCESS	329
53.	SKETCH OF A CASE OF PERIDUODENAL ABSCESS	338
54.	DIAGRAM OF THE PHYSICAL SIGNS OF PYO-PNEUMOTHORAX	
55.	DIAGRAM OF THE PHYSICAL SIGNS OF PERIGASTRIC ABSCESS	348

PART I

PATHOLOGY AND ETIOLOGY

CHAPTER I

MORBID ANATOMY

The term 'simple ulcer of the stomach' is restricted to cases where there is a circumscribed loss of substance of the mucous membrane of the stomach, which extends for a variable depth through the coats of the viscus, but is not attended by any morbid growth. It does not apply to the superficial erosions which so frequently accompany congestion or inflammation of the organ, nor to the multiple form of follicular ulceration which is observed in persons who have died from tuberculosis and other febrile diseases.

1. Number

It is the custom to speak of an 'ulcer of the stomach' as though the disease was usually solitary, and as if the presence of more than one ulcer in the same organ was a circumstance of considerable rarity. Such a belief is, however, contrary to well-established facts. Rokitanski, in his celebrated treatise upon the anatomy of the disease, distinctly states that among his seventy-nine cases no fewer than seventeen presented two or more open ulcers, while Brinton concluded from his researches that multiple ulcers are found in one-fifth of all cases. The post-mortem records at the London Hospital contain 112 cases, in eighty of which the ulcer was solitary, while in the remaining thirty-two it was multiple. In the following table will be found the results of an analysis of 867 cases of open ulcer which we have collected from various sources.

TABLE 1

1 ulcer was pr	resent in	698	cases,	or in	80.5	per cent.
2 uleers were	,,	105	2.3	22	$12 \cdot 1$	***
3 ulecrs		27			3.1	

4 or more ulcers , , , 37 , , 4·26 ,,

It will be observed that in these cases no distinction is made between recent or 'acute' ulcers and those of a chronic character; but inasmuch as these two varieties of the disease are quite distinct from one another, both as regards their



Fig. 1.—Acute ulcers (a, a, a) in the stomach in a case of enteric fever. (London Hospital Museum.)

pathology and their clinical features, it is advisable that an attempt should be made to determine the relative frequency of multiple ulceration in each form of the complaint. In the 112 cases examined at the London Hospital the general appearance presented by the ulcers is stated with sufficient accuracy to permit of a conclusion being drawn as to their probable duration.

Of this number, seventy-one, or 63 per cent., were examples of chronic ulcer, and thirty-three, or 29 per cent., of the acute type of the disease, while in the remaining eight cases (about 7 per cent. of the entire number) a recent perforating ulcer was found to coexist with one of old formation. In only nine out of the seventy-one instances of chronic ulcer was the disease found to be multiple, and in none did the sores exceed four in number. On the other hand, no fewer than eighteen out of the thirty-three cases of acute ulcer presented more than one open sore, while in several their number varied from three to eight. It is evident, therefore, that while chronic gastric ulcer is solitary in about 87 per cent. of the cases in which it occurs, the acute variety exhibits a multiplicity in more than half (54 per cent.).

In rare instances the number of ulcers present in the stomach is excessive. Thus Berthold mentions a case where thirty-four deep perforating ulcers were found in the same organ, while Lange has recorded one in which the inner surface of the viscus was studded with such an infinite number of ulcers that it was impossible to count them. A very similar condition of the stomach was observed a few years ago at the London Hospital in a man who had died of ancurism of the coeliac axis.

Duodenum.—Ulcer of the duodenum is usually solitary. Thus, in the cases we have analysed, a single ulcer was present in 86 per cent., two ulcers in 9 per cent., and three or more in 5 per cent.; while, according to Morot, a single ulcer is found in 81.8 per cent., two in 9.2 per cent., and more than three in 4.5 per cent. Multiple ulcers almost invariably belong to the acute type of the disease, but it is not uncommon to find a recent ulcer situated on the side of the bowel opposite to one of long standing.

2. Size

A gastric ulcer of recent formation is usually small in size, but when the disease has continued for a length of time it may involve a considerable portion of the organ. The acute perforating ulcer which occurs in young adults generally varies from the size of a threepenny piece to that of a sixpence. In rare instances it does not exceed the dimensions of a split pea,

while occasionally it attains the size of a florin. When the disease is associated with dilatation of the heart or cirrhosis of the liver fatal hæmatemesis may ensue from an ulcer near the cardia no larger than a hempseed (fig. 3); indeed, in one case which came under our observation, it was only by injecting the venous plexus around the lower end of the æsophagus that it was possible to detect the site of the hæmorrhage. In other instances the ulcer may possess a diameter of an inch or more without eroding a vessel of importance. In cases of



Fig. 2.—Photograph of a portion of the stomach of a young girl, showing an acute perforating ulcer. Natural size. (Museum of the Royal College of Surgeons.)

multiple ulceration, the sores are usually small and more or less uniform in size.

Chronic ulcers of the stomach often attain very large dimensions. Thus, Cruveilhier mentions an ulcer which measured $6\frac{1}{2}$ inches long by $3\frac{1}{2}$ wide, and Law one which was 6 inches Habershon obby 3. served the whole surface of the stomach between the pylorus and the cardia occupied by a single ulcer, and we ourselves have seen

almost the whole of both surfaces of the organ destroyed by the disease (fig. 4). This exceptional condition is more often due to the coalescence of several separate ulcers than to the circumferential growth of a single sore, and it consequently presents a markedly irregular or crenate edge. In the so-called 'horseshoe' ulcers, as also in those which occasionally surround the pyloric or cardiac orifice, the tract of ulceration is usually broader on one surface of the viscus (posterior) than on the other.

Duodenum.—Like its homologue in the stomach an ulcer of the duodenum varies greatly in size. In the acute variety it is usually small, and often does not exceed the size of a

small pea or a threepenny piece and is seldom larger than a shilling; while the chronic ulcer is much more extensive, and



Fig. 3.—Photograph of a stomach, showing a minute ulcer near the cardiac orifice, which gave rise to fatal hemorrhage. A glass rod has been passed through the branch of the coronary artery which was eroded. (London Hospital Museum.)

may exceed a crown piece in diameter, or may even involve several inches of the bowel.

3. Situation

Numerous inquiries have been instituted with the view of determining the usual situation of a gastric ulcer, and although their results agree in the main, there is yet a considerable difference between the various estimates which have been made concerning the relative liability of different regions of the stomach to the disease. The paramount difficulty which has always beset statistical inquiries upon this subject arises from the absence of any uniform system employed in recording the

locality of an ulcer. Thus while some pathologists recognise only two equal divisions of the stomach, the cardiac and the pyloric, others confine these terms to those parts of the viscus which are immediately contiguous to the two orifices, and make use of the curvatures as intermediate landmarks,

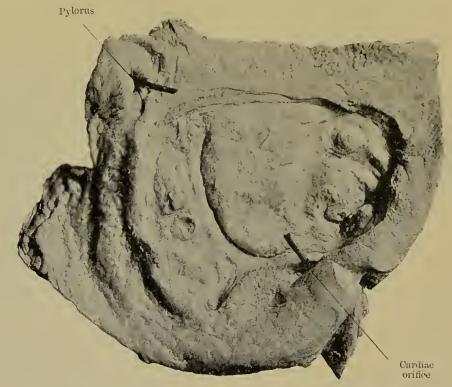


Fig. 4.—Photograph of a stomach, showing an immense chronic ulcer, which extended from one orifice of the organ to the other, and involved the greater part of both surfaces. Much reduced. (London Hospital Museum.)

while others again merely designate the surface of the stomach upon which the ulcer occurred and its distance in inches or centimetres from the nearest orifice. Notwithstanding these difficulties, a careful study of a large number of autopsies clearly indicates that certain parts of the stomach are exceptionally prone to suffer from ulceration, while other regions present an almost complete immunity from the disease. The following table shows the situation of 1,015 ulcers, which we have collected from various sources, and also affords a com-

¹ These include the works of Rokitanski, Lebert, Starcke, Jaksch, Chambers, Habershon, Eppinger, Steiner, Berthold, Wrany, Wollmann, Moore, and Stoll, as well as many of our private cases and those recorded at the London Hospital and London Temperance Hospital.

PUBLO CHIRURGICAL SUCIE ! 1 MORBID ANATOMY 7

parison with the conclusions arrived at by Weleh from a study of 793 cases of the disease. Although it is not noticed in the table, the great majority of the ulcers which occurred near the lesser eurvature were also situated on the posterior wall of the stomach and nearer the pylorus than the cardia.

Table 2.—Analysis of 1,015 Cases of Gastric Ulcer, showing the Relative Frequency of the Disease in different Regions of the Stomach

Position	n			No. of cases	Per cent.	Welch
Pylorus				158	15:6	12%
Lesser curvature				366	36	36.3%
Posterior surface		,		254	25	29.6%
Cardia . ,				80	7.9	6.3%
Great curvature.				42	4.14	3.4%
Anterior surface			. 1	82	8	8.7%
Fundus	,			33	3.3	3.7%

It will be observed that in nearly 76 per cent. of all cases the ulcer is situated in the pyloric region of the stomach near the lesser eurvature and on its posterior surface. It must be remembered, however, that up to the present time no distinction has been made between the aeute and chronic forms of gastric ulceration; and since the latter is by far the more common and striking variety of the disease, it is not improbable that most of the collected cases were examples of this type. It is necessary, therefore, to have recourse to statistics of a more special kind before the relative frequency with which the various regions of the stomach are attacked by each variety of simple ulcer can be indicated with any degree of precision. In the records of the London Hospital we find 109 cases in which sufficient details are afforded for this purpose; of this number seventy were instances of the chronic uleer and thirty-nine of the acute form of the disease. In order to locate the various cases as closely as possible, we have divided the stomach into three imaginary zones, termed the cardiae, middle, and pyloric, by perpendicular lines drawn from equidistant points on the lesser eurvature. When the various ulcers are relegated to their respective situations upon this scheme, the following results become apparent. Of the seventy cases of chronic ulcer, fifty-three occupied the pylorie, seven the middle, and ten the cardiac zone of the organ. Further, in sixty-three cases where additional details are given, forty-six were situated on the

posterior surface, seven on the anterior surface, and ten in the immediate neighbourhood of the lesser curvature, and usually on the posterior wall. It is obvious from these figures that about three-quarters of all chronic ulcers of the stomach are situated in the pyloric region, and on the posterior surface of the organ, and are usually nearer to the lesser than to the greater curvature of the stomach—a conclusion which so closely agrees with the general results expressed in Table 2 as to confirm the supposition that most of the cases it contained were examples of the chronic disease.

When the various cases of acute ulcer are examined in a similar manner, a striking difference is at once apparent. In thirty-nine fatal cases of this disease the ulcer was found in the pyloric zone in thirteen, in the centre of the organ in fourteen, and in the cardiac region in twelve, while in nearly two-thirds of the cases where accurate details are given, the sore was situated in close proximity to the lesser curvature. It is evident, therefore, that the acute ulcer does not exhibit the same proclivity for the neighbourhood of the pylorus as was observed in the case of the chronic ulcer, but develops with almost equal frequency at any spot between the cardia and the pylorus along the upper margin of the stomach, and more frequently on the posterior than on the anterior surface.

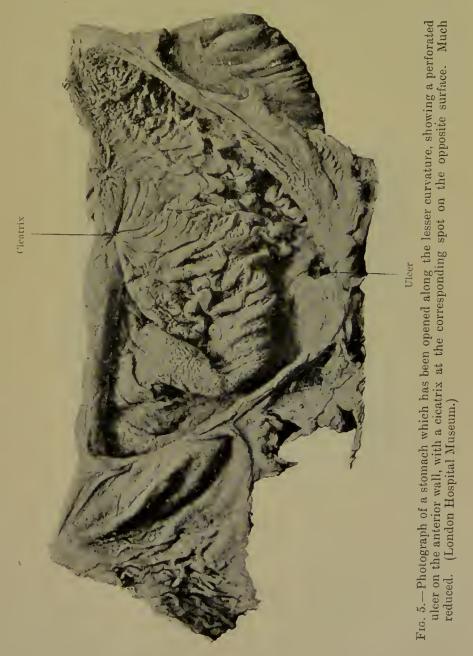
TABLE 3.—SHOWING THE RELATIVE FREQUENCY OF ACUTE AND CHRONIC ULCERS IN DIFFERENT REGIONS OF THE STOMACH

				1	Acute ulcers [39]	Chronic ulcers [70]
Pyloric zone .					13 [33·3%]	53 [75.7%]
Middle zone .	•	•	٠	. ($14 [36\%] \\ 12 [30\%]$	7 [10%] 10 [14%]
Posterior surface	•			: 1	10 [36%]	46 [73%]
Anterior surface.				. /	5 [18%]	7 [11%]
Lesser curvature	•	•	•	• 1	23 [59%]	10 [16%]

Although ulceration is so common along the upper border of the stomach, it rarely occurs exactly upon the line of the lesser curvature, but is usually situated upon one or other surface of the organ within an inch from the attachment of the small omentum.

When two acute ulcers occur in the same stomach, they are usually situated opposite one another on either side of the lesser curvature; but in most cases it can be observed that the

ulcer on the anterior surface is of more recent formation than that on the posterior wall, even when it has produced complete perforation of the coats of the organ (fig. 5). In multiple ulceration the fundus and central portion of the viscus are



usually more affected than the pyloric region. In such instances the disease is very seldom limited to one surface, so that the case recorded by O'Rorke, where six ulcers were present upon the anterior surface, is one of exceptional interest.

Evidences of former ulceration in the shape of puckerings or eleatrices are frequently found in the stomach after death from acute perforating ulcer, and afford an explanation of the acute dyspeptic symptoms from which the patients had suffered at intervals prior to the fatal attack. These sears often require a careful search for their detection, since, being thin and comparatively superficial, they are easily obscured by the rugæ of the mucous membrane. In one case, after escaping the notice of the pathologist, the cicatrix was accidentally discovered by the aid of photography (fig. 6).



Fig. 6.—Acute perforating ulcer of the stomach in a girl. Photograph, natural size. To the right of the ulcer is a small cicatrix, which was discovered by photography. (London Temperance Hospital.)

Sears denoting the presence of previous ulceration of an acute type are most common on the posterior surface of the organ, near the lesser curvature, and not infrequently occur in the immediate vicinity of the open ulcer. They are also found in the fundus, where healing appears to take place with exceptional rapidity. On the other hand, they are rarely encountered on the anterior surface of the stomach, owing probably to the fact that ulcers in this situation have a special tendency to fatal perforation. When acute ulceration accompanies cancer of the stomach, it is usually situated in the immediate neighbourhood of the morbid growth.

Chronic ulceration sometimes involves one of the orifices of the stomach, either by primary implantation or by a process of extension. The former condition is rare, and is chiefly encountered at the esophageal opening; the latter is most common at the pyloric aperture. Ulceration of the cardiac orifice was noted in four of the ten cases (40 per cent.) of chronic ulcer in the cardiac third of the stomach examined at the London Hospital, and in two of these it had produced obstruction to the entry of food into the organ.



Fig. 7.—Crescentic ulcer embracing the cardiac orifice of the stomach. Photograph, natural size. A glass rod is inserted into a branch of the coronary artery. (London Hospital Museum.)

Involvement of the pyloric orifice was noted in eleven out of the fifty-three cases (20.7 per cent.) of chronic ulceration in this region of the stomach, but in almost every instance the disease had commenced at some little distance from the aperture. It would therefore appear that chronic ulceration of the cardia, though comparatively rare, is about twice as often followed by implication of the neighbouring orifice as ulceration of the pylorus, a fact which has hitherto been overlooked.

When two chronic ulcers occur in the same stomach they are usually situated close together on the posterior wall, and in the pyloric region of the organ. If chronic ulceration exists near both orifices, the disease of the cardia has usually followed that of the pylorus. It is rare to find two chronic ulcers on opposite sides of the organ, as so frequently occurs in the acute form of the disease. In about 8 per cent. of the cases, a chronic ulcer is accompanied by one of more recent origin. This secondary ulcer varies in its position, being sometimes situated on the anterior wall opposite the older sore, sometimes near the lesser curvature upon the same surface, while occasionally it occurs at the opposite end of the stomach. There is one position in particular which possesses a considerable degree of interest, both from a clinical and pathological point of view. In several cases of chronic ulcer of the pylorus, with dilatation of the stomach, which have come under our observation, the patient was suddenly attacked by fatal collapse, and in each instance perforation of the stomach was found to have taken place at the lesser curvature close to the cardiac orifice. In one instance a large superficial circular ulcer was found in this position, but in the other cases the tissues presented a blackened and pulpy appearance, with perforation of the organ. This condition was obviously due to extensive hæmorrhage into the coats of the stomach, and was easily distinguished by its general characters and position from the results of post-mortem digestion, while the destruction of the peritoneal tunic, and the subsequent escape of the gastric contents into the peritoneal cavity, afforded an ample explanation of the sudden. abdominal pain and collapse observed during life.1 As the result of some experiments which we conducted in the postmortem room, we found that when the stomach is over-distended with air or fluid there is a tendency for the mucous coat to rupture close to the inner side of the esophageal opening, so it is possible that in these cases of dilated stomach the weakest portion of the organ may have succumbed to the strain, and given rise to sub-mucous hæmorrhage and consequent destruction of the coats of the viscus.

Duodenum.—The usual seat of the ulcer is in the first part of the gut close to the pylorus, which it often involves. Thus

¹ See also cases by Siebert and Andral.

out of the 149 cases analysed by Perry and Shaw, 123, or 82 per cent., were situated in the first part; sixteen, or about 11 per cent., in the second part; and two, or about 1.5 per cent., in the third part: while in the remaining eight cases, or 6 per cent., all three portions of the duodenum were affected. It will therefore be seen that the disease is practically confined to the portion of the bowel above the papilla.

4. General Appearance

A. SHAPE

Gastric ulcers are usually round or oval in outline, and so characteristic is this shape that the disease is often termed 'the round ulcer of the stomach.' It is, however, in the acute form of the complaint that the circular form is most constantly encountered, for in chronic cases a great variety of shapes may be observed. Thus out of seventy-one cases of chronic ulcer

examined at the London Hospital, sixty-one are described as 'round' or 'oval,' two as 'quadrilateral,' one as 'triangular,' two as 'linear' or 'oblong,' and the remaining five as 'irregular' in outline.

Occasionally a chronic ulcer presents a notched or crenated edge owing to an irregular extension of Fig. 8.-Chronic ulcer of triangular shape.

The discussion or in some Photograph, reduced. (London Hospital cases, to the coalescence



Museum.)

of several neighbouring sores. In all these deviations from the circular form the greatest diameter of the ulcer lies obliquely, or even at right angles, to the long axis of the stomach, and its direction of extension is consequently parallel to the principal branches of the gastric arteries.

'Annular' ulceration of the stomach is always chronic in character, and was observed in nearly 6 per cent. of the cases which we have collected. Two varieties may be distinguished,

the 'complete' and the 'incomplete.' The first occurs in the form of a band, which encircles one of the orifices of the stomach, usually the cardiac. During its progress the disease frequently involves the muscular sphincter, and causes contraction of the aperture. In rare instances an annular band of ulceration surrounds the pyloric end of the stomach at a distance of two or three inches from the orifice.\(^1\) The 'incomplete' variety, or 'horseshoe' ulcer, as it is often termed, is usually encountered near the centre of the viscus, which it



Fig. 9.—Chronic ulcer with an irregular edge; probably caused by the fusion of two sores. Photograph, natural size. (Museum of the Royal College of Surgeons.)

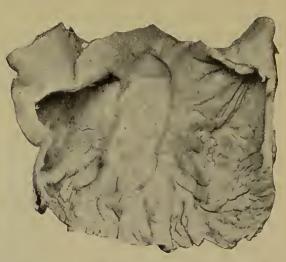


Fig. 10.—Photograph of a stomach showing a large chronic ulcer, which partially encircles the pyloric end of the organ. Much reduced.

embraces for about two-thirds of its circumference. Commencing upon the posterior wall, where it is always deepest and most extensive, the disease passes directly across the lesser curvature, and, gradually becoming more superficial and tapering, proceeds over the anterior surface towards the great curvature (fig. 10).

These ulcers frequently cicatrise, but in so doing they contract to a remarkable degree, and divide the stomach into two

¹ In a case recorded by Dickinson the pylorus was also involved by the ulcer.

pouches, between which only a minute aperture may eventually remain (hour-glass stomach). Sometimes the cicatrisation is incomplete, and the posterior part of the ulcer continues open long after the remainder has healed and contracted the stomach. 'Horseshoe' ulcers not infrequently terminate by perforation of the coats of the stomach, and in such cases the perforation is almost invariably found upon the anterior surface, at the spot where the disease is actively progressing.

The exact cause of the circular form exhibited by the majority of gastric ulcers has been the subject of some discussion. The mode of distribution of the terminal branches of the gastric vessels affords a sufficient explanation of the round shape possessed by those varieties which originate from vascular occlusion, but it does not throw much light upon the problem in the case of chronic ulcers, or in those which arise from catarrhal affections of the mucous membrane. As the result of some experiments which we performed some years ago, it was found that the administration of dry sulphate of zinc to dogs was often followed by well-marked ulceration of the stomach. When the animals were killed within twenty-four hours the mucous membrane of the cardiac region of the organ was seen to be studded with sloughing patches of various sizes and shapes. When sufficient time was afforded for the exfoliation of the necrotic tissue the resultant ulcers rapidly assumed a circular shape, so that, by regulating the times at which the various animals were killed, it was possible to observe the gradual conversion of a linear or oblong abrasion into an ulcer of definite circular shape. We have also watched the same phenomenon in gastrostomised animals after mechanical injuries had been inflicted upon the mucous membrane, but in these cases it was necessary to implicate the muscular tissue of the stomach and to adopt measures to prevent rapid healing of the abrasion. It appears to us, therefore, that the circular shape which is commonly assumed by ulcerations of the stomach is capable of a more simple explanation than is usually accorded to it. The mucous coat of the stomach is a highly elastic membrane which is spread uniformly over the smooth muscular layer with the interposition of only some loose connective tissue. On the principle of the equalisation of tension any perforating wound of such an clastic surface must of necessity assume a circular shape, and

in the present case such a tendency would be aided by the eonstant and varied movements of the stomach which ensue from the contraction of its longitudinal, circular, and oblique muscular fibres. It is interesting to observe, in support of this theory, that in every case of irregular-shaped gastric ulcer the edges of the sore are so indurated and thickened as to be incapable of retraction, while not only is the submucous tissue condensed and firmly adherent both to the mucous and muscular layers, but the movements of the organ itself are usually trammelled by the adhesion of the base of the ulcer to some fixed organ in its vicinity. The origin of the annular variety of ulcer is more difficult of explanation, except in these eases where it can be shown to arise from the fusion of contiguous sores. It is probable, however, that in most cases it eommences by a process of subacute or chronic ulceration on the posterior surface of the stomach near the lesser curvature, and subsequently enlarges along the course of some diseased branch of the coronary artery.

Duodenum.—As a rule the ulcer is circular or oval in shape, especially when of recent origin. In the chronic variety the disease often acquires a quadrilateral form, or it may encircle the bowel and involve the greater portion of the circumference of the gut. The acute ulcer usually exhibits the punched-out appearance with which we are familiar in the stomach, but in the chronic stage the walls are sloping, terraced, or funnel-shaped, with irregular, inverted, or everted edges. Deeply excavated ulcers with overhanging edges are by no means uncommon.

B. EDGES AND BASE

The edges of the Acute Ulcer, as observed on the postmortem table, are sharply defined and clean-cut. As the ulcer penetrates the coats of the organ it destroys each successive layer of tissue to a lesser degree than the one above it, so that the final opening in the peritoneum may be only from one-third to one-sixth of the size of the aperture in the mucous membrane. The result of this peculiar mode of invasion is to give to the ulcer the general appearance of a truncated cone or funnel, the base of which is situated at the mucous surface, while the apex corresponds to the peritoneal aspect of the disease. In most cases the sides of the cone are placed somewhat obliquely to the surface of the mucous membrane, so that its apex, instead of being situated centrally, deviates slightly to one or other side of its median axis. When the ulcer is situated near the lesser curvature, the lower wall slopes so that the apex points upwards; while in those rarer instances where the disease occupies the lower segment of the stomach the apex of the cone is directed downwards towards the great curvature of the organ. This conical aspect of the disease is most conveniently studied in cases of multiple ulceration; for when death has ensued from perforation of a single sore the ulcer usually appears as if it had been 'punched out' of the wall of the stomach, owing to post-mortem contraction of the muscular tissue and to the tearing of the softened peritoneum during the removal of the stomach from the body.

The walls of a recent ulcer are invariably soft and free from any sign of inflammatory thickening, thus offering a marked contrast to the tough induration which characterises the chronic form of the disease. In those of very recent formation the walls and floor are often bright red, and either infiltrated with blood or dotted over with small blood-clots and tags of necrotic tissue. In other cases the mucous and muscular tissues are slightly yellow or pale grey in colour, while in those which have existed for some time the colour of the edges and base of the sore does not differ materially from that of the surrounding mucous membrane. Œdematous swelling of the walls, with the formation of a yellow tumid ring at the circumference of the ulcer, is occasionally observed when the disease has originated during the course of enteric fever, pyæmia, or anthrax.

The base of an acute ulcer may be formed by the submucous, muscular, or even by the peritoneal coat of the stomach. In very rare instances the liver, pancreas, diaphragm, colon, mesentery, or the abdominal wall may constitute the actual floor of the sore, being closely united to it by recent adhesions. The peritoneal aspect of the disease varies according to the depth of the ulcer, but it usually shows signs of capillary injection as soon as the necrotic process has invaded the deeper layers of the muscular coat, and is sometimes covered with recent lymph. In those cases which terminate rapidly in perforation the actual rupture of the stomach is usually very minute, and presents the appearance of

a small ragged aperture or vertical chink which may be closed with lymph. On the other hand, when perforation occurs from the rupture of recent adhesions around the base of the ulcer, the aperture may be as large as that in the mucous membrane, and the same effect is often produced by the careless removal of the stomach from the body.

Such are the main features presented by a recent ulcer on the post-morten table, but there are many reasons for believing that they convey but a very inadequate picture of the disease as it exists during life. Since Beaumont first published his observations upon the naked-eye appearances of the living stomach in its various phases of health and disease, the fact that post-mortem changes invariably obliterate the most striking evidences of gastric inflammation has been well established, though it is seldom remembered. The vivid redness and tumefaction of the mucous membranc. with its pustular excrescences, local hæmorrhages, and turbid secretion, which Beaumont observed during an attack of gastric catarrh so mild as to produce merely a general malaise in the patient, have but a sorry counterpart in the opaque, leaden-looking, or, at most, slightly congested tissue which is observed at an autopsy, even in cases where death is directly attributable to inflammation of the stomach. If we then acknowledge the astonishing transformation in appearance which the mucous membrane undergoes as soon as life is extinguished, is it possible to believe that an acute ulcer of the stomach mcrely exhibits during life the appearance of a 'punched-out hole' which the after-death picture has imprinted upon our minds? or is it conceivable that an abrasion of the inner surface of the stomach not exceeding a threepenny piece in size can give risc to the violent pain and vomiting which so rapidly ensue from the introduction of a little food into the organ? Any one who has been accustomed to experiment with the living stomach, or who has watched the formation of an artificially induced ulcer through a gastrostomy wound, can testify to the extraordinary congestion and swelling of the mucous membrane which occur at the seat of disease, and which are often so profound as to completely obscure the primary Icsion. In this connection it is interesting to note that, in more than one instance where laparotomy has been performed for perforation of the stomach, a very similar condition has been

observed. Thus, in a case published by Dickinson and Haward, the following statement occurs: 'A gaping perforation of the stomach was found, of the size of a threepenny piece. Around this perforation, which was obviously the apex of a simple ulcer, the gastric wall was thickened over an area fully as large as the palm of a hand; and by reason of this thickening it was impracticable to invert the gastric wall so as to close the perforation by sutures.' Nevertheless at the autopsy on this case it is recorded that 'the thickening round the perforation observed at the operation had disappeared.' We have also known a case where the presence of a diffuse swelling in the region of the pylorus, which was discovered upon abdominal section, caused the surgeon to give a diagnosis of cancer of the stomach, although at the autopsy a few days later a simple ulcer the size of a shilling was all that remained in the place of the supposititious tumour. It appears to us, therefore, that instead of picturing an acute ulcer as a small hole in the gastric wall surrounded by a zone of congested vessels, we should accustom ourselves to the conception of a large, acutely inflamed patch in the stomach, the tissues of which are sometimes so swollen and ædematous as to give a sense of brawny induration to the touch, while the ulcer itself is almost obscured from view on the inner surface by the congestion and tumefaction of the surrounding mucous membrane.

The cause of the conical shape which is exhibited by many recent ulcers has given rise to considerable discussion. The explanation which finds the widest acceptance is base upon Virchow's theory concerning the embolic origin of the disease, for there is no doubt that any sudden obstruction of a small artery of the stomach is followed by a wedge-shaped infarct in the mucous membrane, the base of which is situated at the surface, and the apex near the peritoneal coat. It will be shown, however, when the pathogenesis of the disease is discussed, that primary vascular obstruction, though an occasional, is by no means a constant, cause of gastric ulceration, and consequently the peculiar shape of the ulcer must be susceptible of some other explanation. In this connection the following experiments are of some interest.

Experiment 1.—A stomach removed from a rabbit immediately after death was well washed and placed in a warm

and moist chamber, under which conditions it retained its power of muscular contraction for a considerable time. A small cylindrical platinum cautery was heated to a degree considerably below redness, and the point applied to the mucous membrane. In about a minute the whole thickness of the stomach was burnt through, and the organ was then replaced in the warm chamber. On examining the lesion after a short interval, the orifice on the mucous aspect was found to present a somewhat greater diameter than the cautery, the edges being raised and tumid from contraction of the muscularis mucosæ. But the perforation in the muscular coat appeared to become progressively smaller as the peritoneal surface was approached, while the external orifice would no longer admit the point of the cautery. In this manner a funnel-shaped perforation had been produced by a cautery of equal diameter throughout.

Experiment 2.—Abdominal section was performed upon a guinea-pig, and three small threads, which had been previously soaked in a solution of chromic acid and afterwards dried, were inserted through the wall of the stomach, in the cardiac, middle, and pyloric zones respectively. In the course of the next forty-eight hours the animal succumbed to acute general peritonitis. At the autopsy it was found that the irritation of the thread in the fundus had given rise to a circular perforation the size of a threepenny piece, but that the thread in the pyloric region remained grasped by the muscular tissue. Upon removing it and opening the stomach, the mucous membrane presented a circular ulcer the size of a split pea, the aperture in the deeper structures being distinctly funnel-shaped.

It would seem, therefore, that in many cases the acute perforating ulcer tends to assume a conical shape owing to the contractile properties of the tissues subjacent to the mucous membrane. The gastric juice, while it dissolves that portion of the muscular tissue immediately opposed to it, irritates the fibres at the circumference, and hence each successive film of muscle tends to contract and thus to minimise the extent of its injury. In this manner the actual area of tissue offered to the solvent action of the gastric juice becomes progressively smaller, until by the time the peritoneum is invaded the possible damage to its structure is comparatively insignificant.

This hypothesis is confirmed by the fact that it is only in the pyloric region, where the muscular tissue is thick, that an ulcer acquires the typical funnel shape.

The Chronic Ulcer is distinguished from the acute form chiefly by the appearance of its edges and base; since, after the disease has existed for some time, reactive inflammation takes place in the surrounding mucous membrane, with the result that the coats of the organ in the immediate vicinity



Fig. 11.—Simple chronic ulcer of the stomach, with an oval perforation in its base. Photograph. (London Hospital Museum.)

of the sore become sclerosed and firmly adherent one to the other.

The surface of the stomach around the ulcer usually shows signs of chronic inflammation, being considerably thickened in texture, brown or slate-grey in colour, and not infrequently covered with small polypoid vegetations. In cases of subacute ulceration, or when the chronic process has become temporarily progressive, the surrounding mucous membrane is greatly congested and tumid in appearance, and may exhibit minute extravasations of blood or a few hæmorrhagic erosions on its

surface. Another condition which is worthy of notice is the occasional occurrence of varicose veins in the neighbourhood of a chronic ulcer. Either these may form a ring round the sore at a little distance from its edge, or one or more varices may occur at isolated spots. When partial cicatrisation has taken place it is not unusual to find a large thin-walled vein at the junction of the scar and the healthy mucous membrane, which, as healing proceeds, becomes gradually enlarged and dragged towards the centre of the cicatrix. This dilated condition of the superficial venules arises from the pressure exerted upon the large trunks lying in the submucous tissue by the contraction of the newly formed fibrous tissue round the ulcer, and constitutes a twofold source of danger; for not only does fatal hæmorrhage sometimes ensue from the rupture of the varix, but the local congestion to which it gives rise retards the process of healing, and may even produce secondary ulceration of the scar.

The edges of the chronic ulcer vary greatly in their general appearance according to the activity of the disease. In a typical case the ulcer exhibits in a marked degree the conical or crater-like formation previously described. At one side of the sore the edge is slightly elevated, smooth, hard, and perpendicular, while the opposite wall presents a sloping or terraced appearance as it descends gradually towards the base of the ulcer.

When the coats of the stomach are unusually thick, it sometimes happens that the muscular structures retract, and thus undermine the mucous membrane for a considerable To such an extent is this sometimes carried that the edges of the mucous membrane may meet in the centre of the aperture, and almost screen from the view the large globular cavity which exists in the subjacent tissues. In callous ulcers of long standing, the edges of the sore are often elevated above the level of the surrounding mucous membrane, and, being hard, irregular, and everted, may so closely resemble an epithelioma of the stomach that microscopic examination is necessary before its benign nature can be definitely established. have even known this mistake to be made during life, owing to the fact that the hard ulcer could either be felt through the abdominal walls, or was discovered by an exploratory incision. Very chronic ulcers situated near the pylorus often exhibit the most curious distortions from irregular contraction

of their edges. One of these abnormalities consists of a kind of cave in the substance of the organ, the mouth of which is flush with the surface, while the roof is composed of mucous membrane.

When the process of healing commences, the edge of the ulcer becomes smooth, regular, and slightly depressed, while the walls slope evenly and gradually down towards the base. In many instances the various processes of extension and repair may be observed in the same ulcer at different parts of its margin.

The floor of the ulcer is composed either of one or more coats of the stomach, or of some neighbouring organ which has



Fig. 12.—Chronic ulcer of the stomach, with sloping edges. Photograph, much reduced. (London Hospital Museum.)

become adherent to it. In the former case, the base of the sore is usually smooth, and consists chiefly of bundles of muscular fibres; but occasionally it presents a series of narrow terraces or ridges, or it is distinctly hummocky in appearance. If the disease is indolent in type, the edges and base are usually smeared over with a thick brown or greenish secretion; but if the sore has been recently enlarging, its activity is betrayed by a soft, shaggy, or gelatinous appearance of the tissue, or by the presence of small sloughs and clots of blood. True granulations are rarely observed, although Müller has recorded a case

where this phenomenon was encountered, and we have also met with an instance of a similar character. It is by no means uncommon to observe one or more thin white cords, which are either embedded in the walls of the ulcer or are partially enclosed in its floor. These cords are branches of the gastric arteries which normally ramify between the deeper coats of the stomach, and which have undergone obliteration owing to inflammatory thickening of their coats and subsequent coagulation of the blood contained in them. When death has ensued from hæmorrhage, the ruptured vessel is usually found to be exposed at only one spot on the floor of the ulcer, and to have escaped this safeguarding process of endarteritis and thrombosis. In rare instances, small aneurisms are found upon the sides of a vessel which has lost its normal support by the destruction of the surrounding tissues, and fatal hæmatemesis has been known to occur from the rupture of one of these minute diverticula (Powell, Sachs, Welch, Rasmussen).

When the floor of a chronic ulcer is composed entirely of some neighbouring organ to which it has become adherent through the medium of local peritonitis, the coats of the stomach are found to have undergone complete destruction, and the adherent viscus fills the aperture like a plug. If the tissues of the adjacent organ are naturally tough, as in the case of the pancreas, the chronic inflammation which is induced in them produces a condition of fibrosis which gives the base of the sore the appearance and consistence of fibrocartilage.

In certain cases, however, either this protective inflammation is defective in character or the newly formed tissue fails to resist the solvent action of the gastric juice, with the result that the floor of the ulcer becomes gradually excavated. Thus cases have been described where large cavities were produced in the liver, spleen, and pancreas; while Rokitanski has recorded a case in which a piece of the latter organ became completely detached, and was found free in the cavity of the stomach. On the other hand, the soft and thin-walled organs, like the duodenum, colon, and diaphragm, are seldom able to offer a stiff backing to the ulcer, which consequently perforates their coats and establishes a fistulous communication between the stomach and the adjacent cavity.

The peritoncal surface of a non-adherent chronic ulcer is

invariably much thickened and condensed from chronic inflammation. In such cases perforation usually occurs near the apex of the ulcer close to its abrupt edge, and results from the detachment of a small fragment of the serous membrane which had been previously converted into a yellow slough.

Microscopical Appearances.—An examination of the edge of a recent ulcer shows the disease to be sharply defined at its upper part, while in the deeper portions of the mucous membrane the gastric glands appear to have been abruptly

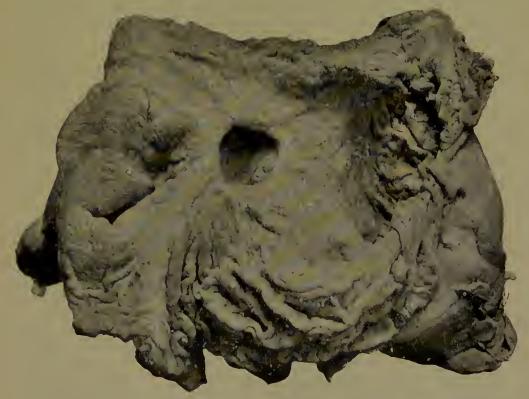


Fig. 13.—Chronic ulcer of quadrilateral shape on the posterior wall of stomach, which had formed a deep excavation in the substance of the pancreas. Photograph, much reduced. (London Hospital Museum.)

cut off in their descent towards the muscularis mucosæ. The tissues immediately surrounding the edges of the sore are more or less infiltrated with blood, the red cells of which occasionally preserve their normal outlines, but for the most part appear distorted in shape, disintegrated, and mixed with swollen connective-tissue elements and non-staining nuclei. This detritus is particularly abundant in the interstitial connective tissue of the mucous membrane, and presses upon and displaces the gastric tubules so that they can scarcely be distinguished one

from another in the lower two-thirds of their extent. Where, however, the glands can be distinctly seen, they are found to be swollen and irregular in outline or filled with blood corpuscles and granular débris. The submucous and muscular coats show similar signs of infiltration in the neighbourhood of the ulcer, though in a lesser degree. The capillary vessels are distended with blood, and occasionally small hyaline thrombi may be recognised in the arterioles near the base of the ulcer (Recklinghausen).

If the ulcer is examined at a later period, the zone of molecular disintegration is found to have disappeared, while the edges of the sore either are quite clean or at most present a few tags of necrotic tissue or minute clots of blood. The tissue immediately surrounding the disease shows indications of a slight reactive inflammation in the form of a round-cell exudation between the fundi of the tubules. The ducts of the glands are dilated, and their orifices blocked with mucus, nuclei, and disintegrated blood-cells, while the solitary glands in the neighbourhood are somewhat swollen.

A section of a chronic ulcer shows that the induration of the edge which characterises the disease is due to the formation of fibrous tissue. The connective tissue which normally supports and separates the gastric glands is greatly thickened and often fibrillated in the vicinity of the ulcer, so that the superficial portions of the tubules are either completely obliterated, or twisted and diverted from their perpendicular course. fundi of the glands often exhibit a cystic appearance from retention of their secretion, the central cells being replaced by either cubical or columnar epithelium, or degenerated and partially detached from their basement membrane. Korczinski and Jaworski have met with four cases of chronic ulcer in which there was well-marked hyperplasia of the parietal cells of the glands similar in character to that which often accompanies hypersecretion of the gastric juice. The various coats of the stomach around the ulcer are firmly united to each other, but in spite of this the muscular coat often retracts and displaces the structures of the mucous membrane, so that under the microscope the mouths of the glands around the disease seem to point towards the cavity of the ulcer. This simple phenomenon has led Witosowski to conceive the fantastic idea that the activity of gastric ulceration is due to the constant

dripping of the secretion from these distorted glands on to the sides and base of the sore. Such a theory requires no serious discussion, inasmuch as the oblique position of the glands is obviously an accidental result of the ulcer, while the secretory epithelium of the tubules themselves is invariably so altered by disease as to be incapable of furnishing any active juice. The submucous tissue around the ulcer is greatly thickened and condensed by the organisation of the abundant roundcell exudation, while in many cases the muscular coat is almost completely destroyed by the compression exercised upon its structures by the interstitial formation of fibrous tissue. The arterioles which ramify in the deeper part of the mucous coat and in the connective tissue beneath it, usually present evidences of chronic inflammation of their inner and middle coats, which in some instances gives rise to complete occlusion of their lumina (Cornil and Ranvier). Occasionally the arteries and veins situated near the base of the ulcer are either blocked with blood clot or filled with thrombi of a hyaline character. Openchowski has also noted the occurrence of hyaline changes in the walls of the vessels themselves. Thickening and degeneration have likewise been observed in the nerves distributed to the mucous membrane in the neighbourhood of the disease.

CHAPTER II

THE RESULTS OF ULCERATION

1. Adhesions

As an ulcer continues to perforate the gastric wall, the peritoneum which lies immediately in the course of the advancing necrosis becomes inflamed and covered with a thin layer of lymph. If the disease occurs in a part of the stomach which is comparatively stationary and habitually in contact with some solid organ, the inflamed base of the ulcer is apt to adhere to the latter, and should no accident arise to disturb the relative position of the parts, a firm union eventually takes place between them. Such adhesions differ in no respect from those met with in other parts of the peritoneal cavity, except that, owing to the chronicity of the ulcer, they are often very dense and tough. Thus it may be impossible to separate the wall of the stomach from the pancreas, liver, or diaphragm, owing to the firm fibrous tissue which has welded the two structures together, and which is often so hard as to turn the edge of the knife. Occasionally lime salts are deposited around the base of the ulcer, giving to the adhesions the character of mortar.

It is usually supposed that every chronic ulcer of the stomach sooner or later contracts adhesions with some neighbouring organ, but this belief is in excess of the truth. Jaksch found adhesions in twenty-two out of the fifty-seven cases he examined (40 per cent.), Lebert in forty-one cases out of ninety-eight (42 per cent.), while at the London Hospital adhesions are mentioned as having been present in thirty-three out of seventy-one cases (46 per cent.) of chronic ulcer, a result which agrees very closely with the conclusions arrived at by the aforementioned writers. On the other hand, among the thirty-three examples of acute ulcer which occur in the

same records, firm adhesions were only noted twice. The following table, which has been compiled from an analysis of 123 cases of gastric ulcer accompanied by adhesions, shows the relative frequency with which the various organs in the vicinity of the stomach become attached to the base of the ulcer.

TABLE 4

Organ						Number of cases adherent	Percentage
Pancreas (alone)						49	40)
Liver (alone).						33	26.8 74.8%
Pancreas and liv						10	8.1)
Colon						7	5.7
Liver and colon						4	$3\cdot 2$
Spleen						2	1.6
Mesentery .						3	2.4
Three or more of	rgans	1 .				15	12.2
						123	100

It will be observed that in nearly 75 per cent. of the cases the ulcer had contracted adhesions either with the pancreas or the left lobe of the liver, or with both of these organs. The remarkable frequency with which these viscera are affected obviously depends upon their close proximity to the ordinary situation of the ulcer, since it has been estimated that nearly 76 per cent. of all chronic ulcers of the stomach occur in the pyloric region, and on the posterior surface. A little consideration will also show that the nearer the ulcer is to the centre of the posterior wall the greater will be the chance of its adhesion to the underlying pancreas; while the closer it is situated to the lesser curvature the greater the likelihood of its union with the liver. Ulcers near the centre of the lesser curvature, especially if they occur on the anterior wall of the viscus, almost invariably become adherent to the left lobe of the liver, and those which develop near the cardiac orifice not infrequently become attached to the diaphragm also. Ulcers situated on the anterior wall below its upper third have comparatively little opportunity of contracting firm adhesions, owing to the great mobility of this region of the stomach, and to the absence of stability in the tissues with which it comes into contact. Occasionally,

¹ Including pancreas, liver, diaphragm, abdominal wall, colon, spleen, omentum, mesentery, kidney, and right adrenal.

however, union occurs with the eolon, aided by thickening of the great omentum, which is sufficiently close to allow the formation of a bi-mucous fistula. The spleen is rarely affected, owing to the infrequency of ehronic ulceration in the fundus of the stomach. Cases have been recorded, however, where fatal hæmorrhage has occurred from erosion of the splenic pulp.

Duodenum.—In sixty-two cases of duodenal ulcer where the necessary details were given, we find that adhesions existed

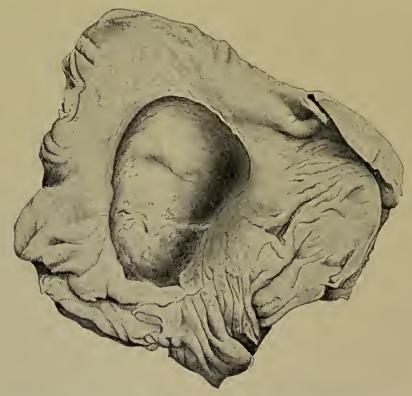


Fig. 14.—Drawing of the posterior wall of a stomach, showing a large chronic ulcer, through which the pancreas projects in the form of a tumour. (London Hospital Museum.)

between the base of the sore and some neighbouring organ in twenty-six, or 42 per cent. When the disease attacks the anterior wall of the bowel, fatal perforation usually ensues, owing to the absence of any solid viscus which can serve as a secondary base for the uleer; but when it affects the posterior aspect of the gut, perforation is often prevented by the panereas, which lies immediately behind it. If it were not for the presence of this organ, the vast majority of acute ulcers of the duodenum would give rise to perforation. Ulcers situated upon the

upper border of the first portion occasionally contract adhesions with the gall-bladder, or with the quadrate lobe of the liver, while in rare cases disease of the second or third part of the bowel becomes adherent to the stomach, colon, small intestine, aorta, or spinal column. Out of the twenty-six cases in which adhesions were present, the pancreas was affected in twenty-one, the gall-bladder in three, and the liver in two instances.

The formation of adhesions around the base of an ulcer must be regarded as an attempt on the part of nature to prevent sudden perforation of the stomach, and, failing this, to minimise the extent of the mischief by localising the subsequent peritonitis. The achievement of this latter object becomes manifest in cases of 'perigastric' or 'subdiaphragmatic' abscess, where the area of suppurative peritonitis is strictly limited by the fibrous adhesions which have previously formed in the neighbourhood of the disease. It might also be said that even when an ulcer occurs in those regions of the stomach where there is no solid organ to serve as a support, the existence of adhesions serves to guide the perforation in the direction which is least dangerous to life. This is seen in the formation of fistulæ after attachment of the base of an ulcer to the colon, abdominal wall, or diaphragin.

Unfortunately, most natural benefits have also inherent drawbacks, and even peritoneal adhesions are by no means an unmixed blessing. It will be shown hereafter that the mere union of the base of an ulcer to a fixed organ like the pancreas or liver, constitutes one of the prime obstacles to its healing, since its fixation renders it incapable of undergoing, the contraction which is requisite for repair. Again, a considerable proportion of the deaths from hæmatemesis result from the erosion of some neighbouring viscus which has become adherent to the base of the ulcer. Thus the vessels of the pancreas and liver are frequently opened with fatal consequences, while in certain cases the portal vein, the splenic pulp, and even the heart itself, have been perforated. Adhesions may also be responsible for the occurrence of acute general peritonitis. Thus cases have been recorded where a sudden muscular effort, such as sneezing, coughing, vomiting, or straining at stool, has caused the base of an ulcer which was

firmly adherent to the liver or diaphragm to be torn through, and the contents of the stomach to be distributed over the surface of the peritoneum. Finally, it may be mentioned that adhesions frequently displace and distort the stomach, tranmel its movements, and interfere with the functional activity of the neighbouring viscera.

2, Plastic Perigastritis

It occasionally happens that, instead of the peritoneal inflammation remaining localised to the immediate neighbourhood of the ulcer, it exhibits a tendency to progressive extension, so that eventually the greater part of the surface of the stomach is involved in the inflammatory process. This diffuse variety of perigastritis occurs quite independently of perforation, and is more commonly associated with ulceration of the cardia than of the opposite end of the stomach. The disease is very chronic in its character, and gives rise to considerable thickening of the peritoneal aspect of the stomach, of the great omentum, and of the peritoneal folds which connect the organ with the liver and spleen. As the result of this the stomach becomes firmly adherent to the liver, spleen, diaphragm, abdominal wall, pancreas, and colon, and in many cases the matting together of these organs is so intense that it is impossible to isolate the viscus from the surrounding structures. When the ulcer is situated on the posterior wall the peritoneal inflammation may be limited by the lesser omentum and the transverse mesocolon, so that the posterior aspect of the viscus becomes closely united to the pancreas, spine, and the other structures behind the abdominal cavity. In either case the result is to hamper the movements of the stomach, while in the diffuse form of the disease the contraction of the thickened peritoneum may so compress and distort the organ as to reduce it to the size and shape of a piece of bowel.

Another, but less common, result of the plastic inflammation is the formation of fibrous cords which pass across the anterior surface of the stomach between the liver and the great omentum. In one case of this kind which was examined at the London Hospital, a twisted band was found that not only divided the stomach into two unequal pouches, but had also

given rise to chronic ulceration of the mucous membrane along the line of compression. Cirrhosis of the liver, perihepatitis, or perisplenitis is sometimes associated with the peritoneal mischief, and in rare cases tubercular peritonitis eventually supervenes.

3. Cicatrisation

A. FREQUENCY OF SCARS IN THE STOMACH

The natural tendency of all simple ulcers of the stomach is to undergo repair. Experiments upon the lower animals have conclusively proved that abrasions of the inner wall of the stomach, from whatever cause they may have arisen, rapidly heal; indeed, a chronic gastric ulcer can only be produced with the greatest difficulty and by the adoption of special precautions. The same fact is observed after injuries have been accidentally inflicted upon the human stomach. Thus cases have been recorded in which large pieces of the mucous membrane have been torn away by the careless use of the stomach-pump, . or where the viscus has been injured by the swallowing of knives and other sharp instruments; nevertheless within a very short time these injuries were completely healed. Again, an acute ulcer of the stomach which has penetrated sufficiently deep to cause severe hæmatemesis is a common complaint in early adult life; but when such cases are properly treated there is no reason to doubt, from the subsidence of the symptoms and their non-recurrence, that the mischief has undergone complete repair. It is, however, from the study of morbid anatomy that this tendency to spontaneous cure is brought most prominently before us.

The discovery of a well-marked scar in a stomach affords unmistakable evidence of former ulceration, and consequently an inquiry into the frequency with which such cicatrices are found on the post-mortem table is of great value in determining the proclivity of gastric ulceration to spontaneous repair. Brinton found that the autopsies made by Dittrich, Jaksch, Willigk, and Dahlerup revealed a total of 147 scars and 156 ulcers, 'making the proportion of the former nearly equal to the latter;' while Fox concluded from his researches that

A somewhat similar case is reported by Robinson. Path. Trans. vol. iv. p. 134.

cicatrices are found nearly twice as often as ulcers. From a more extended study of published statistics Welch believes that it would be a moderate estimate to place the ratio of cicatrices to ulcers at 3 to 1. It must always be remembered, however, when recourse is had to statistics of this nature, that, while open ulcers never fail to attract attention, small cicatrices are easily overlooked unless the stomach is specially examined for them. In other words, the more attention devoted to the subject, the greater will be the number of healed ulcers discovered. This view is supported by statistics which have been compiled at Prague and Copenhagen. At the former place the percentage of cicatrices found in the stomachs of persons dying of different diseases varied under successive pathologists (Jaksch, Dittrich, Willigk, Eppinger) between 0.81 and 2.44; while at the latter Grünfeld found that his original estimate of 11 per cent, became increased to 20 per cent, when extra care was bestowed upon the examination of the stomach.\(^1\) Out of 729 cases we have collected with reference to this point, we find that an open ulcer was present in 249 and a cicatrix in 480; a ratio of about 1 to 2. These cases, however, appear to have been chiefly of the chronic type, and the scars were probably large enough to attract immediate notice, so that the figures throw no light upon the real frequency with which ulcers of the stomach undergo spontaneous cure. Considering how much more common acute ulcers are than those of the chronic form, and how rarely death results from them, we think it must be conceded that at least four out of every five simple ulcers of the stomach heal during life.

The number of scars encountered in the same stomach does not constitute an inquiry endowed with any practical importance. It may be mentioned, however, that out of 370 cases where the cicatrices were carefully counted, we find that

1 scar existed in 72·1% 2 scars , , , 12·3% 3 ,, ,, , , 5·6% 4 or more scars in 10%.

When these results are compared with those expressed in Table 1, it will be seen that multiple cicatrices are slightly more frequent than multiple ulcers.

Gastric ulcer appears to be extremely prevalent at Copenhagen. (See Etiology.)

Duodenum.—Owing to the thinness of the wall of the bowel an ulcer of the duodenum is far more apt to terminate fatally than its homologue in the stomach, and for this reason scars are comparatively infrequent. According to Perry and Shaw cicatrisation occurs in only about 11 per cent. of the cases, but since the superficial scars which result from the acute form of the disease are easily overlooked it is probable that this estimate is somewhat below the mark. Cicatrices have been found within a month of the infliction of a severe burn (Curling, Holmes).

B. CHARACTERS OF THE CICATRIX

If the ulcer has not penetrated deeply into the coats of the stomach, or if it has undergone a rapid cure, the site of the

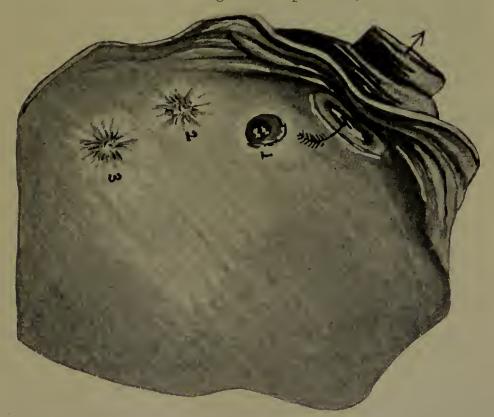


Fig. 15.—Sketch of the cardiac end of the stomach, showing an acute ulcer (1) and two superficial scars of former ulcers (2 and 3). Natural size (Museum of the London Temperance Hospital.)

injury is marked by only a small white spot in the mucous membrane. These little scars are often overlapped and

concealed from view by the neighbouring ruge, so that their detection is a matter of considerable difficulty, and it may be necessary to stretch the stomach in front of a bright light before they can be recognised with certainty. When, however, the ulcer has been more extensive, the site of the former disease is marked by a dull white, opaque scar of stellate form, which is slightly depressed below the surface, and surrounded by radiating folds of thickened mucous membrane. These cicatrices may often be recognised on the exterior of



Fig. 16.—Cicatrix of a chronic ulcer. Photograph, natural size. (Museum of the Royal College of Surgeons.)

the stomach by a puckered and contracted appearance of the peritoneum.

A large ulcer often remains imperfectly cicatrised near its centre owing to the arrest of the healing process. Cases of this kind are very prone to relapse from the breaking down of the thin scar-tissue surrounding the open sore, and not infrequently fatal hæmorrhage occurs from the erosion of a vessel at the one open spot of an otherwise wellhealed ulcer (Cruveilhier).

Occasionally the cicatrix is surrounded by a zone of dilated veins, some of which may eventually rest upon the scar itself. At an autopsy which we recently witnessed upon a case of fatal hæmorrhage, the bleeding was found to have arisen from the rupture of a large vein which crossed the centre of a healed ulcer. In rare instances the scar exhibits the characters of keloid. In a case of this kind which came under our notice the cicatrix formed an oblong mass which projected nearly three-quarters of an inch above the surface of the mucous membrane. Microscopical examination showed that the mass was composed of fibrous tissue.

See also a case cited by Leith, Allbutt's System of Medicine, vol. iii. p. 443.

C. THE PROCESS OF CICATRISATION

Small gastric ulcers produced artificially in animals are repaired by the formation of genuine peptic glands derived from the superficial epithelium which grows over the surface from the edges of the wound, so that the new tissue closely resembles the normal mucous membrane in structure and appearance (Griffini and Vassale.) This probably occurs also in cases of hæmorrhagic erosion and other slight varieties of ulceration in the human subject, since such abrasions of the inner surface of the stomach heal without visible scars; but when the ulceration is more extensive, repair is brought about by the contraction of newly formed fibrous tissue which gradually approximates the edges of the sore. Ulcers of large size, and those which have contracted adhesions with some solid organ like the pancreas, are unable to undergo the degree of contraction that is necessary for the approximation of their edges. In these cases the surface heals by the formation of a dense scar-tissue, which remains permanently uncovered by mucous membrane. Cicatrisation, however, is often imperfect, and the ill-nourished scar-tissue is very prone to fall a prey to secondary ulceration, with the result that the disease alternately heals and breaks out afresh.

Microscopical examination of the edge of a healing ulcer shows an abundant exudation of round cells in the various tissues. In the mucous coat they are most noticeable near the muscularis mucosæ, where they surround the fundi of the gastric glands and penetrate the interstitial tissue in all directions. In the submucous coat the inflammatory exudation is most marked in the neighbourhood of the small arteries and veins, while in the muscular tissue the small cells congregate between the bundles of fibres and in the subperitoneal tissue.

Subsequently this round-cell exudation is succeeded by the connective-tissue elements which characterise the final cicatrix. The gastric tubules in the margins of the ulcer undergo a series of changes of considerable interest. According to Hauser these glands present at first all kinds of degenerative changes, being broken, compressed, and widely separated from one another by the thickened interstitial tissue, or converted into small cysts. When healing has progressed for some

time, an active proliferation of the deeper portions of the remaining tubules may often be observed, the fundi lengthening and branching in all directions, both into the submucosa and also into the fibrous base of the uleer. These tubular processes are lined either with cylindrical cells or with flat cpithelium, and on section present the general appearance of an adenoma. They are supposed to be the starting-point of cancer when it attacks a scar in the stomach.

4. Changes in the Shape of the Stomach

The contraction of an uleer, with or without eleatrisation, is apt to give rise to alterations in the shape of the stomach, the nature and degree of which depend upon the size and situation of the disease. These morbid conditions are as follows:—

- (a) Dilatation of the stomach.
- (b) Contraction of the stomach.
- (c) Irregular contraction, with approximation of the two orifices.
 - (d) Hour-glass deformity.
 - (e) The formation of pouches.
- (a) Dilatation of the stomach frequently follows chronic ulceration, and may arise from three distinct causes:—
- 1. Almost every ease is associated with a moderate degree of gastreetasis owing to the eoexistence of chronic gastric catarrh. In these cases the ulcer may be situated at any part of the organ, and need not interfere with the patency of the pyloric orifice. The mucous membrane around the disease is usually thickened and slate-grey in colour, and is not infrequently studded with small polypoid vegetations. Elsewhere the inner surface of the stomach is either swollen and opaque or superficially congested. Under the microscope, the characteristic features of chronic parenchymatous and interstitial inflammation are easily recognised. Clinically, the viscus seldom extends below the level of the umbilieus, periodic vomiting is absent, while the ordinary symptoms of gastric catarrh are often accompanied by those of hypersecretion.
- 2. The adhesion of an uleer near the pylorus to the under surface of the liver has the effect of suspending the organ at one spot, with the result that its peristaltic movements become

ment of the viscus. This condition leads to fermentation of the gastric contents and secondary catarrh of the mucous membrane, which in their turn aggravate the tendency to gastrectasis by further enfeebling the contractile power of the muscular coat. From these various causes the stomach becomes gradually dilated until it may rival the size which the organ attains in cases of pyloric stenosis. It is also probable that the drag which the bulky viscus exercises upon the fixed pylorus when the patient is in the erect position, causes a further obstacle to the passage of food into the bowel by kinking the first portion of the duodenum. That this is not a mere fanciful suggestion is evidenced by the spontaneous subsidence of vomiting which often follows the maintenance of rest in the recumbent posture.

3. The most important variety of dilatation of the stomach is due to obstruction of the pyloric orifice by an ulcer in its vicinity. In these cases the viscus may attain an enormous size, and completely fill the anterior aspect of the abdominal cavity. The appearance of its walls depends upon the degree of stenosis: when the obstruction is moderate the muscular coat of the organ is usually hypertrophied and the mucous membrane thrown into heavy folds; but if the contraction of the pylorus is extreme and of long standing, the parietes of the stomach become thin and transparent, the ruge on the inner surface are obliterated, and the mucous membrane of the fundus shows signs of atrophy.

The degree of stenosis varies greatly in different cases. As a rule, the aperture is reduced to only two-thirds or one-half of its normal size, but in rare instances it may be so contracted as to barely admit the passage of a probe. Moderate stenosis was noted in nine out of our fifty-three cases of chronic ulcer in the pyloric third of the stomach, but in only two cases was the obstruction extreme. Brinton computed that severe stenosis occurs in one out of every 200 cases of gastric ulcer. Although it is impossible to offer any absolute opinion upon this point, it is probable that the pylorus becomes partially obstructed in 16 to 20 per cent. of all chronic ulcers situated in its vicinity, but that in only about 2 per cent. of these is the stenosis extreme. Complete obliteration of the orifice is very rare, even as a result of the destruction

of the mucous coat of the stomach by corrosive fluids. Only one instance of the kind (hydrochloric acid poisoning) has come under our personal observation, and in this case the stomach was contracted rather than dilated, owing probably to the incessant vomiting which had occurred during life.

Gastric ulcer may produce obstruction of the pylorus, either by constricting the orifice or by causing its displacement. The former condition is by far the more common, and the actual narrowing is usually due to inflammatory thickening of the mucous and submucous tissues around the ulcer, whereby the communication between the stomach and the bowel, instead of being a mere aperture, is converted into a narrow canal. Occasionally the mucous membrane is thrown up into an irregular fold in front of the orifice in such a manner as to form a valvular obstruction to the exit of the gastric contents. In one of our cases enormous dilatation of the stomach ensued from this interesting condition.

In other cases the contraction of the edge of an ulcer situated near the pylorus converts the circular aperture into a triangular or oblong slit, and thus produces a serious diminution of its diameter. Small cicatrices in the immediate neighbourhood of the valve may thus occasion the same degree of stenosis as a large ulcer more remotely situated. In two out of our fifty-nine cases of pyloric ulceration the disease was situated upon the aperture; but although the stomach was greatly dilated in both instances, the orifice itself presented hardly any sign of stenosis. It must therefore be inferred that muscular spasm plays an important part in the production of gastrectasis when the sphincter is involved in the ulcerative process.

Displacement of the pylorus with consequent obstruction of its orifice occurred in only three out of the fifty-nine cases. In one instance an ulcer was found to have twisted the pylorus upwards and inwards so as to bend the stomach upon itself. In another the disease had contracted adhesions with the second part of the duodenum, thereby throwing the pylorus upwards and kinking the first portion of the bowel. Thickening of the small omentum with adhesion of the ulcer to the gall-bladder and peripyloritis were responsible in the third case for the obstruction.

(b) Diminution in the size of the stomach may arise from the plastic perigastritis already referred to, but it is usually the

result of a constriction of the cardiac orifice by a chronic ulcer in its vicinity. Among the 112 cases of gastric ulcer examined at the London Hospital, stenosis of the cardia was noted in two instances, while Starcke met with a similar condition in one out of his thirty-nine cases of the disease.

It is usually stated that while pyloric stenosis is a frequent result of gastric ulcer, occlusion of the cardia from the same cause is very rare. That pyloric obstruction is by far



Fig. 17. - Chronic ulcer, embracing the cardiac orifice of the stomach. Photograph, natural size. A glass rod is inserted into a branch of the coronary artery. (London Hospital Museum.)

the more common of the two is beyond a doubt, but this is easily accounted for by the fact that about 75 per cent. of all chronic ulcers are situated nearer to the pylorus than to the cardia. On the other hand, if the cases of ulceration of the cardiac end of the stomach are considered separately, it is found that stenosis of the orifice is by no means an infrequent result of the disease. Thus contraction of the aperture was observed in two out of our ten cases of chronic ulcer situated in the cardiac third of the organ, or in one-fifth of the total number. It is obvious, therefore, that stenosis of the cardiac orifice occurs quite as often from ulcers situated

in the cardiac third of the stomach as obstruction of the pylorus from disease in the pyloric zone.

The manner in which the stenosis is produced is essentially the same as that described in the case of the pylorus. In most cases the ulcer occupies the lesser curvature immediately to the inner side of the orifice, with the result that when it cicatrises the aperture becomes distorted and displaced. In other instances the sphincter itself is involved in the ulcerative process, so that its muscular structure becomes gradually replaced by fibrous tissue. In rare cases the formation of a perigastric abscess displaces the cardia and prevents the entry of food into the viscus. It has already been noted that ulceration of the pyloric sphincter may give rise to obstruction of the orifice by exciting muscular spasm. This variety of functional stenosis is even more frequently observed in cases of ulceration of the cardia, owing probably to the constant irritation of the sore by the passage of food. In two of our cases, where the ulcer involved the orifice and the patient exhibited the clinical phenomena of esophageal stricture during life, no contraction of the aperture could be discovered at the autopsy (see fig. 17). Cardiospasm must therefore have been responsible for the fatal obstruction.

Simple ulceration of the œsophagus just above the stomach is an occasional cause of stenosis of the cardia. Frerichs observed a case where an ulcer of this description had produced extreme cicatricial contraction of the lower end of the œsophagus, and two similar instances have been recorded at the London Hospital, in one of which the pylorus, and in the other the duodenum, also presented an open ulcer.

The actual size of the stomach varies with the severity and duration of the stenosis. When the obstruction to the entry of food has only been slight, the viscus may maintain its normal dimensions; but if the disease has been sufficiently pronounced to produce immediate regurgitation of the food, the stomach is usually much reduced in size. A remarkable example of this recently came under our notice in a man who for several weeks had been unable to take any form of nourishment. At the autopsy the stomach was found retracted under the left hypochondrium, and closely resembling a cricket-ball in appearance. On section its cavity appeared to be almost

¹ See also cases by Quincke, Zahn, and Wilks.

obliterated, and its walls thickened. Under the microscope, however, the coats of the viscus were found to be quite healthy. This extraordinary condition was the result of a simple chronic ulcer which involved the cardiac orifice without seriously contracting it, the symptoms of stenosis observed during life being probably due to muscular spasm.

(c) The contraction of a chronic ulcer situated upon the lesser curvature may produce a curious deformity of the stomach, the salient feature being the approximation of the two orifices. In cases of medium severity one or two inches may still



Fig. 18.—Photograph of a stomach, showing extensive scarring from former ulceration. The whole organ was much contracted, and the two orifices only about two inches apart. (London Hospital Museum.)

intervene between the esophageal and pyloric apertures (fig. 18), but if the whole or greater part of the upper border of the viscus has been the seat of disease the two orifices may become practically bound together by fibrous tissue. In such cases the organ is usually adherent to the under surface of the liver, and presents the appearance of a lady's purse-bag, owing to the pleating of its anterior surface. Occasionally the stomach becomes greatly dilated from the displacement and consequent obstruction of the pylorus, while in some cases secondary

ulceration occurs in the fundus or in the vicinity of the scar. This variety of deformity was observed in two out of our ten cases of chronic ulcer on the lesser curvature, and in one instance was associated with a large subphrenic abscess.

(d) Hour-glass deformity of the stomach is a rare but important result of chronic ulceration. It usually arises from the contraction of a horseshoe or girdle ulcer, but a similar appearance may also be produced by the cicatrisation of several small ulcers near the lesser curvature on the opposed surfaces of the organ. In this deformity the stomach appears to be divided into two pouches by a constriction about its centre, the upper and larger sac being formed by the fundus and the lower and smaller one by the pyloric portion of the The line of division between the two shows itself externally in the form of a deep groove which varies in width from one-eighth to three-quarters of an inch, and is usually situated somewhat nearer the pylorus than the cardia. On its posterior and upper aspect the constriction is firmly adherent to the pancreas and under surface of the liver. When the stomach is opened the cardiac pouch is found to consist of the dilated fundus, the walls of which are thinner than normal and whose mucous membrane is devoid of rugæ. The pyloric pouch, on the other hand, is seldom dilated; its walls are often thickened and its various coats adherent to each other from chronic inflammation. The channel of communication varies considerably in diameter; in some cases admitting the forefinger with ease, while in others an ordinary lead pencil can scarcely be passed through it. In all cases the cause of the deformity is readily observed in the dull white scar which practically encircles the stomach at the affected spot, and from which puckered folds of mucous membrane radiate in all directions. Not infrequently, owing to the incomplete healing of the original ulcer, a callous-looking sore is found at the spot where the stomach is adherent to the pancreas or liver, while occasionally one or more ulcers of recent formation may be observed in the immediate neighbourhood of the stricture. These latter probably owe their origin to irritation of the gastric mucous membrane produced by the retained food. The occasional formation of a bilocular stomach by the pressure of fibrous adhesions between the liver and the omentum has already been described in the section dealing with plastic perigastritis (page 32).

45

THE RESULTS OF ULCERATION

It was formerly believed that hour-glass deformity of the stomach was usually due to the contraction of an ulcer, but the researches of Carrington and others appear to show that this supposition is an erroneous one. In about 45 per cent. of the cases which have been recorded, neither ulcer nor scar could be detected in the stomach, while in the great majority of the cases where an ulcer was present it was obviously of more recent formation than the stricture. It is also to be observed that in the congenital disease both the stricture itself and the two divisions of the organ are much more regular in outline than in the acquired form, while the adhesion of the stomach to the neighbouring organs, which is an almost invari-

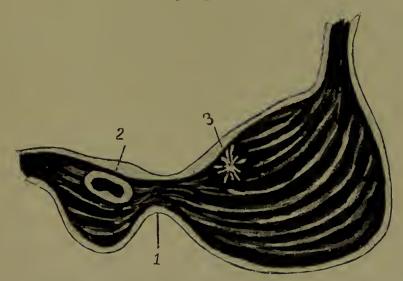


Fig. 19.—Hour-glass stomach of congenital origin. 1, site of the constriction; 2, large chronic ulcer in the pyloric region; 3, scar of a former ulcer. (From a specimen at the London Temperance Hospital.)

able feature of the latter complaint, is never encountered in the former. That the deformity is a rare result of ulceration is proved by the fact that only one case of the kind is mentioned in the records of the London Hospital for forty years, whereas several instances of the congenital form of the disease were encountered during the same period of time.

Duodenum.—A healing ulcer of the duodenum may also give rise to a kind of hour-glass deformity in which the pylorus forms the point of constriction between the dilated stomach on the one side and the bowel on the other. Thus in one of our cases where an annular cicatrix was found in the second part of the duodenum, both the portion of the gut above it and the

stomach were enormously dilated, while the pyloric orifice measured nearly one inch and a half in diameter. In another case the duodenal stenosis was associated with a contracting ulcer at the lower end of the œsophagus which had produced fatal inanition. In this instance, both the stomach and bowel were greatly reduced in size. Biermer relates a case where obliteration of the lumen of the duodenum was followed by dilatation of the æsophagus. In rare cases cicatricial stenosis of the duodenum is associated with a girdle ulcer of the stomach, so that the organ appears to be divided into three sacs of unequal size (V. Hacker). Adhesions of a chronic ulcer to the pancreas, liver, or gall-bladder may give rise to curious distortions of the gut.

If the ulcer is situated over the papilla, the process of healing may obliterate the opening of the biliary and pancreatic ducts, and lead to great dilatation of these channels. Rupture of the gall-bladder has been known to follow its abnormal distension from this cause (Herzfelder).

(e) Pouches.—It might readily be supposed that ulceration of the stomach might so weaken the wall of the viscus as to permit bulging or pouching of the tissues at the affected spot, in the same way that the wall of a diseased artery will often dilate from the pressure of its contained blood. As a matter of fact, however, the formation of pouches or diverticula in the stomach from this cause is very rare, probably on account of the inflammatory thickening of the base of the ulcer, and its frequent adhesion to some solid organ in its neighbourhood.

Among the various recorded cases of gastric diverticula we have been able to find only one instance where the pouch appears to have been produced by the stretching and protrusion of the floor of an ulcer. In this instance a large chronic ulcer, which was situated close to the pylorus and invaded the walls of the stomach on either side of the lesser curvature, had undergone cicatrisation and produced contraction of the orifice. In the centre of its floor was a pouch about the size of a cherry, which projected on the outer surface of the stomach and communicated with the cavity of the viscus by a small aperture. The inner surface of this diverticulum was composed of scar-tissue, while the remainder of its wall consisted principally of thin muscle. In all the other recorded eases,

with the exception of one, the diverticulum was situated between the ulcer and the pylorus. Thus Orth relates a case where a large ulcer of annular shape in the pyloric region of the stomach was found to have contracted firm adhesions with the liver, omentum, and colon. Only a small portion of the anterior wall between the ulcer and the pylorus had escaped being involved in the adhesions, and this, being relatively unsupported, had become expanded by the intragastric pressure into a large pouch, the orifice of which was wide enough to admit four fingers. The pouch was lined by thin mucous membrane, and the rest of its wall was composed of strands of muscular tissue and peritoneum. Somewhat similar cases have been recorded by Cruveilhier, Brinton, and Kleine. The only instance of this condition with which we are personally acquainted was recently observed in the post-mortem room of the London Temperance Hospital. As in the preceding cases, a chronic ulcer had become adherent to the liver so as to leave a small portion of the wall of the stomach which lay between the ulcer and the pylorus unsupported. At this spot a large diverticulum had formed, which passed upwards and backwards beneath the liver. The thin mucous membrane with which the pouch was lined presented a catarrhal ulcer which had perforated the sac and induced fatal peritonitis.

In each of these various cases the diverticulum must be regarded as a kind of hernial protrusion of a part of the stomach arising from a lack of support combined with an increased tension of the gastric contents due to stenosis of the pylorus. Kolaczek has recorded a case which proves that a pouch may sometimes arise from direct traction upon the wall of the stomach in the vicinity of an ulcer ('traction diverticulum'). In this instance a large sac developed in the cardiac end of the viscus, owing to the adhesion of an ulcer to the abdominal wall.

Duodenum.—Diverticuia are far more common in the duodenum than in the stomach, and usually occur independently of disease. They are most frequently found between the pylorus and the biliary papilla, and vary in appearance from a slight bulging of the coats of the bowel to a sac the size of an egg. When the pouch is small, its walls are composed of all the coats of the intestine, but when it attains a considerable size its tissues are very thin, and consist of

expanded mucous membrane covered by peritoneum. Such diverticula are merely hernial protrusions of the mucous coat through the muscular tunic, and probably owe their origin to the abnormally high pressure of the intestinal contents between the pylorus and the lower fixed portion of the duodenum. As a rule they possess no clinical significance, but occasionally they form receptacles for the lodgement of gall-stones (Harley), or by penetrating the substance of the pancreas compress the

pancreatic and bile ducts (Rolleston).

When diverticula occur as the result of ulceration they are usually found opposite the seat of discase, or between the ulcer and the pylorus (fig. 20). The presence of a pouch was noted in 6 per cent. of our cases of chronic ulcer.

Occasionally the ulcer itself becomes pouched owing to distension of its base, and in such cases the lining membrane of the sac presents the appearances of fibrous tissue. More rarely a true

'traction diverticulum' is formed by the adhesion of the base of an ulcer to the liver, diaphragm, or colon (Perry and Shaw). These sacs may undergo secondary ulceration leading to perforation or hæmorrhage, or they may compress the biliary and pancreatic ducts or establish a fistulous communication with the colon (Stewart).

Fig. 21 shows the pyloric end of the stomach with the commencement of the duodenum laid open along its outer curvature. Immediately contiguous to the pylorus, on the posterior wall of the intestine, is a deeply recessed ulcer. It forms the floor of a pouch about three-quarters of an inch deep, which projects behind the wall of the duodenum

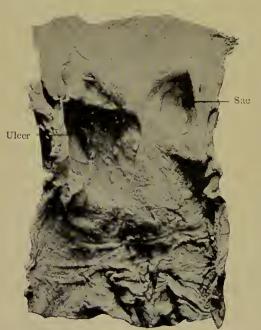


Fig. 20.—Photograph of the first portion of the duodenum, showing a large recessed ulcer with a diverticulum opposite to it. The bowel has been opened along its upper margin. (London Hospital Museum.)

backwards and downwards into the substance of the pancreas. The mouth of the pouch is irregularly quadrilateral in shape, and the mucous membrane is folded over its margins.

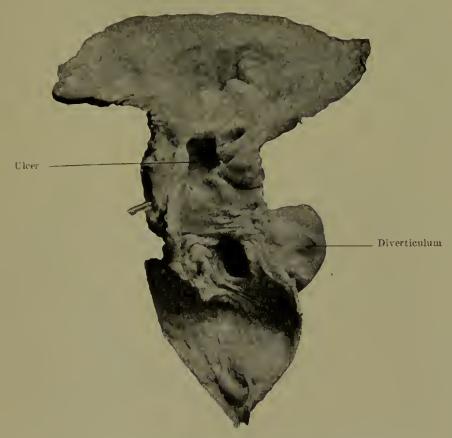


Fig. 21. - Photograph of a duodenum, showing a deeply recessed chronic ulcer just below the pylorus, with a large diverticulum lower down. Slightly reduced. (London Hospital Museum.)

About two inches lower in the duodenum, and contiguous to the other margin of the line of section, is seen a simple pouch formed by a hernial protrusion of the mucous membrane through the muscular wall. The orifice is about the size of a shilling, and the pouch itself measures one inch and a quarter in length, and three-quarters of an inch in width.

5. Erosion of Blood-vessels (Hæmatemesis and Melæna)

It is probable that ulceration of the stomach is invariably accompanied by some degree of hæmorrhage, although it is often too small in amount to colour the fæces or the vomit.

In such cases the blood is derived from the capillary vessels of the surrounding tissues, which are usually much swollen and congested. The more severe forms of hæmorrhage are due to the erosion of a vessel situated in the submucosa, or between the muscular and serous coats of the stomach, while in those rare cases where life is brought to an abrupt termination by profuse and uncontrollable bleeding, the disease is usually found either to have destroyed a large artery or to have invaded the structure of some neighbouring vascular organ. such as the liver, spleen, or heart. Hemorrhage from the stomach is a frequent cause of death in gastric ulcer. Out of the 112 fatal cases recorded at the London Hospital, eighteen, or 16 per cent., died from hæmatemesis, while in another series of 298 necropsies on gastric ulcer we find that fatal hæmorrhage occurred in fifty-six cases, or in 18.8 per cent. It is to be observed that fatal hæmorrhage is chiefly encountered in cases of chronic ulcer, only about 3 per cent. of our 'acute' cases having succumbed to this cause.

The source of the hamorrhage.—Fatal bleeding may ensue from the erosion of (1) an artery, (2) a large vein, (3) a vascular organ, the exact source in each case varying according to the position of the ulcer and the depth to which it has penetrated. A glance at the following diagram of the blood supply of the stomach will show that the principal vessels from which danger is to be apprehended are the coronary and epiploic arteries which traverse the curvatures of the organ, and the splenic vessels which run along its posterior aspect, since the vast majority of chronic ulcers occur in one or other of these situations. Thus Brinton found that in 48 per cent. of his cases of fatal hæmatemesis the ulcer occupied the small curvature, in 34 per cent. the posterior surface, and in 4 per cent. the anterior surface; while out of seventy-four cases which we have collected, the ulcer was situated near the lesser curvature in 54 per cent., near the inferior border in 6 per cent., and on the posterior wall of the organ in about 40 per cent.

This conclusion is confirmed by the results of an inquiry as to the exact vessel found to be eroded in the various cases. In sixty-six cases where the source of the bleeding was accurately noted, a branch of the coronary artery was affected in thirty-two, the splenic artery in fourteen, the right gastro-epiploic artery in six, the gastro-duodenal artery in four, the splenic

vein in three, the coronary veins in three, the left gastro-epiploic artery in two, and the plexus of veins round the cardiac orifice in two.

Venous hæmorrhage usually arises from the rupture of a vein which has become dilated from back-pressure. These varices are most frequent in the fundus of the organ or in the immediate vicinity of the cardiac orifice. In the former situation they are usually due to disease of the spleen or to some congenital abnormality of the gastric vessels, while in

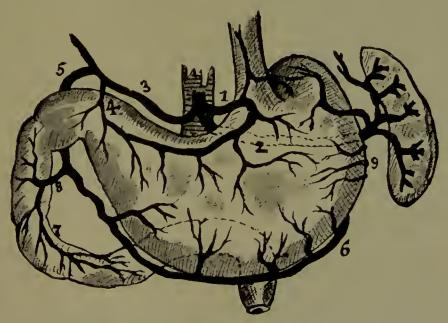


Fig. 22.—Diagram of the stomach and duodenum, showing their arterial blood supply. 1, coronary artery; 2, splenic; 3, hepatic; 4, pyloric; 5, gastro-duodenal; 6, right gastro-epiploic; 7, 8, superior pancreaticoduodenal; 9, left gastro-epiploic.

the latter the cause is to be found in obstruction of the portal circulation, either from heart disease or cirrhosis of the liver.

Owing to the anamic condition of the stomach after death it is often extremely difficult to discover the exact source of the bleeding. Murchison has recorded two cases where a minute pore-like opening in the mucous membrane communicated with a large vessel; and we have found five cases in the records of the London Hospital where an equally minute ulcer had perforated a large superficial vessel with fatal results. Stewart has related a case of fatal hamatemesis in

 $^{^{1}}$ See a case by Lancaster, Clin. Soc. Trans. vol. xxx. p. 32.

a young girl, where a large varix in the fundus of the stomach had been opened by an ulcer three-sixteenths of an inch in diameter. In the neighbourhood of the ruptured varix there were several other large veins, along with a superficial cicatrix.



Fig. 23.—Varicose veins in the stomach, with acute ulceration. Death from hæmorrhage. Photograph, natural size. (Museum of the Royal College of Surgeons.)

The spleen was enlarged and its capsule thickened. There is little doubt that if the stomach and duodenum were searched with greater care 'simple congestion' would very rarely find acceptance as a cause of fatal bleeding from these regions of the alimentary tract.

Occasionally an ulcer in the fundus of the stomach perforates the substance of the spleen, with the result that fatal hæmorrhage ensues from the vessels of the pulp. Cases of this description have been recorded by Jaksch, Steiner, and others. More rarely the vessels of the liver, pancreas, or adrenals suffer in a similar manner. Four cases have been recorded in which a gastric ulcer invaded the substance of the heart and produced fatal bleeding into the stomach and intestines (Oser and Chiari,

Brenner, Finny, Bruenniche).

Duodenum.—Most of the large arteries which supply the duodenum lie immediately behind it or along its inner margin, so that an ulcer has to perforate the muscular coat before it can produce severe hæmorrhage. For this reason almost all the ulcers which give rise to bleeding are very deep and situated upon the posterior wall of the bowel. With regard to the frequency of hæmatemesis and melæna we find that one or both of these symptoms were observed in 26 per cent. of our 'acute' and in 40 per cent. of our 'chronic' cases. In both varieties of the disease the percentage death-rate from hæmorrhage was the same, namely 35. These figures are greatly in excess of the estimate of 13 per cent. arrived at by Perry and Shaw, but closely approximate to those of Krauss, who found that bleeding occurred in 33 per cent. of the cases which he collected. With regard to the source of the hamorrhage it may be noted that out of fifteen cases, the superior

pancreatico-duodenal artery was opened in ten, the gastro-duodenal in three, the pancreatica magna in one, and 'a branch of the splenic' in one. Among the other vessels which have been found affected are the hepatic, pyloric, and coronary

arteries; the inferior vena cava; the portal vein (Frerichs), and the abdominal aorta (Stich). Occasionally a small ruptured aneurism is found upon the exposed vessel (Perry and Shaw).

mechanism of fatal The hæmorrhage. - Considering the wide area of destruction wrought by a chronic ulcer, and the extreme vascularity of the stomach, it seems surprising at first sight that only about 18 per cent. of the fatal cases die from hæmorrhage. It must be remembered, however, that not only is the liability to bleeding minimised to a great extent by obliterative inflammation and thrombosis of the vessels in the immediate neighbourhood of the disease, but that any rent in their walls is rapidly closed by the contraction of the muscular coat and the coagulation of the blood at the seat of puncture. It is probable, therefore, that in every



Fig. 24.—Duodenum in a case of burns, showing a deep ulcer in the first portion of the bowel. A bristle has been inserted into a branch of the pancreatico-duodenal artery, from which fatal hæmorrhage occurred. Photograph, natural size. (London Hospital Museum.)

case of fatal hæmorrhage one or more of the following conditions will be present: (1) Non-obliteration of the vessel previous to its erosion; (2) insufficient contractility of its muscular coat; (3) deficient coagulation of the blood; (4) an abnormally high tension in the arterial or venous system of the organ.

(1) It has already been shown (p. 27) that in many cases of gastric ulcer the arterioles in the immediate neighbourhood

of the disease exhibit a marked thickening of their inner and middle coats, while not infrequently their lumina are completely obliterated by fibrinous or hyaline thrombi. This condition is only met with in cases of chronic ulceration, and is chiefly confined to the small vessels which supply the inucous and submucous tunics of the stomach. These facts explain in great measure the important differences which exist between the hæmorrhages in the acute and chronic forms of the disease. In acute ulcer moderate hæmatemesis is an extremely common symptom, owing to the rapid destruction of the tissues, the vessels of which are unprotected by antecedent thrombosis; but fatal bleeding is proportionately rare, since the ulcer involves only a small area of the stomach, and never extends beyond the serous coat. In chronic ulcer, on the other hand, the small vessels are usually thrombosed before they are involved by the necrotic process, and consequently the disease may exist for many years without obvious bleeding; as soon, however, as the deeper tissues become involved, the large branches of the gastric arteries are apt to become eroded, so that the hæmorrhage when it occurs is always severe and often fatal.

(2) Inefficient contraction of the ruptured vessel may arise from several causes. In many cases the coats of the artery are so thickened by inflammation that they lose their elasticity and power of contraction, while in other instances the vessel, being firmly embedded in the fibrous wall or base of the ulcer, is converted into a rigid tube. This latter condition is especially conspicuous in those cases where an artery has been eroded in the centre of a cicatrix (Cruveilhier, Boullay). Rupture of a miliary aneurism is also apt to be followed by fatal hæmorrhage, owing to the absence of contractile tissue in its walls and to the rigid patency of the vessel from which it springs.

(3) Tardy and insufficient coagulation of the effused blood plays an important part in the causation of fatal bleeding, although the conditions which prevent the formation of a firm clot are not fully understood. It would seem, however, that in many cases severe antecedent anæmia may be responsible for this abnormal state of the blood, while in others it is possible that some other constitutional derangement may give rise to it. In this connection it is interesting to observe that severe hæmorrhage often ensues from the comparatively superficial ulcers

which occur during the course of typhoid, diphtheria, and pyæmia, and is usually a prominent symptom of chronic ulceration in syphilitic subjects. Lastly, it may be mentioned that the dangerous character of venous hæmorrhage probably depends as much upon the deficient clotting of non-aërated blood as upon the inefficient contraction of the thin-walled vein.

(4) An abnormally high tension in the vessels of the stomach is probably a factor of no mean importance in the production of hæmorrhage from a gastric ulcer. Thus, out of our eighteen cases of fatal hæmatemesis, the ulcer was found to occupy the pyloric region in eight, the centre of the organ in two, and the cardiac region in eight. In four out of the eight cases of pyloric ulcer the heart was considerably hypertrophied from Bright's disease, and in each case the hæmorrhage was due to the rupture of an artery. On the other hand, four out of the eight cases of ulceration of the cardia presented signs of mitral disease with chronic congestion of the liver, while in another instance there was also hepatic cirrhosis. It is interesting to observe that in at least two of the cases, where there was congestion of the stomach from obstruction of the portal circulation, the hæmorrhage was due to the erosion of a vein; while in two others the source of the bleeding was apparently not discovered. It is also probable that a sudden increase of blood-pressure in the abdominal organs is responsible for the attacks of hæmatemesis which in some cases of gastric ulcer occur only at the menstrual periods.

6. Perforation of the Stomach

Complete perforation of the coats of the stomach is a frequent result of ulceration, but its effects vary considerably under different conditions. If no adhesions have formed around the base of the sore prior to the accident, the contents of the viscus will find their way into the general cavity of the peritoneum and set up a diffuse suppurative inflammation. On the other hand, if adhesions are present, the inflammation is apt to be strictly circumscribed, and a localised abscess is developed instead of general peritonitis. Lastly, should the base of the ulcer be firmly adherent to some neighbouring organ, like the liver, pancreas, or colon, the only result will be a pro-

gressive necrosis of the new tissue, with the ultimate production of a cavity in its substance or the establishment of a fistula.

(1) Acute General Peritonitis.—The frequency of general peritonitis as a cause of death in ulcer of the stomach has been variously estimated by different writers. Lebert noted it in 12 per cent. of his own cases, and in 37 per cent. of the fatal cases of the disease which he had collected; while in our own series of autopsies this mode of termination was observed in thirty-two out of the 112 cases, or in 28.5 per cent. These latter estimates are somewhat reduced when a larger number of cases is investigated, for we find that in 678 autopsies with open ulcers, perforation had occurred in 153, or in about 22.8 per cent.

Site of the ulcer.—The modern treatment of perforation of the stomach by abdominal section renders it necessary that the usual site of the perforation should be determined as accurately as possible. Brinton was the first to point out that the incidence of perforation does not in any way correspond to the incidence of ulceration; or, in other words, that the common seat of ulceration is by no means the most frequent site of perforation. On the contrary, he found that 70 per cent. of all perforations occur on the anterior surface of the stomach, 21 per cent. on the lesser curvature, and only 9 per cent. on the posterior wall. This exceptional liability to perforation by an ulcer situated on the anterior wall is probably due to two causes. In the first place, owing to the extreme mobility of this part of the stomach and to the absence of any fixed organ in its vicinity, there is no opportunity for the formation of protective adhesions, so that when perforation occurs the contents of the stomach escape directly into the peritoneal cavity. second place, an ulcer on the anterior wall is often unaccompanied by characteristic symptoms, so that the existence of the complaint may escape notice until the sudden occurrence of the fatal accident.

The relative frequency of perforation in different regions of the stomach, as determined by post-mortem evidence, does not coincide with the experience gained by abdominal section during life; for while the pathologist teaches that the pyloric end of the organ is most often the seat of the lesion, the surgeon is convinced that the opening is usually situated near the cardia. This apparent discrepancy is, however, easily

explained when it is remembered that most of the cases which eome to an autopsy are examples of the chronic disease, while those which call for operation almost invariably belong to the acute form in young women. It is also probable that, owing to the difficulty of reaching the cardia through a small abdominal incision, an operator is apt to regard an ulcer as being nearer to the esophagus than is actually the case. In the following table a contrast is offered between the frequency of perforation in various parts of the stomach in the acute and chronic forms of the disease respectively:—

Table 5. An Analysis of 351 Cases of Fatal Perforation, showing the Liability of Different Regions of the Stomach to Perforation in the two Varieties of Gastric Ulcer.

No.			Pylo- rus	Middle	Cardia	Near lesser curva- ture
78	80.5%	19.5%	20%	24%	56%	50%
223	62%	38%	45%	30%	25%	44%
50	36%	64%	62%	26%	12%	60%
	78 223	78 80·5% 223 62%	78 80·5% 19·5% 223 62% 38%	78 80·5% 19·5% 20% 223 62% 38% 45%	78 80.5% 19.5% 20% 24% 223 62% 38% 45% 30%	78 80.5% 19.5% 20% 24% 56% 223 62% 38% 45% 30% 25%

It will be observed that whereas the acute disease usually perforates the comparatively thin coats of the stomach in the eardiac half of the viscus, close to the lesser curvature and on the anterior surface, the chronic form of the complaint is most prone to perforate in the pyloric portion of the organ on the posterior aspect near the upper margin.

In the vast majority of cases the extravasation of the gastric contents is followed at once by an acute suppurative inflammation of the peritoneum, the only exception to this rule being found in those rare instances where the peritoneum is already affected by chronic tuberculosis, in which case the results of perforation of the alimentary tract are less marked (Fenwick). It is possible, however, that there may be other conditions which render the peritoneum comparatively insusceptible to ordinary irritation; for Bardeleben has recorded a case in which, although the patient lived twenty-four hours, and purgative medicines together with the contents of the stomach were found in the cavity of the

peritoneum, no signs of inflammation were discovered after death.

Duodenum.—Perforation of the duodenum leading to general peritonitis is a frequent result of ulceration, owing to the thinness of the wall of the bowel. It was observed in sixty-one out of our 124 fatal cases, or in about 49 per cent. It is exceptionally frequent in that form of acute ulcer which occurs as a primary disease in young men; for out of our twenty-five fatal examples of this disease no fewer than twenty-three, or 92 per cent., died of perforation.

With regard to the situation of the ulcer, we find that in 94 per cent. of the cases it occupied the first portion of the duodenum, and, in the remaining 6 per cent., the second part. The anterior wall, being unprotected by neighbouring viscera, is the usual site of the accident, for it is recorded that the ulcer occupied this position in 86 per cent. and the posterior wall in 14 per cent. of the cases where its exact location was described.

- (2) Perigastric Abscess.—A localised abscess may occur in connection with an ulcer of the stomach under three conditions:

 (1) If adhesions have previously formed around the base of the sore in such a manner as to prevent extravasation of the gastric contents into the general cavity of the peritoneum;

 (2) if the initial leakage from the perforation is so slight as to cause a strictly localised peritonitis which in its turn serves to circumscribe a later and more copious extravasation; (3) if the perforation occurs in certain localities which are outside the peritoneum, as, for instance, between the layers of the lesser omentum or mesocolon, or in the lesser cavity of the peritoneum, the orifice of which (foramen of Winslow) has been previously obliterated.
- (a) Frequency.—A perigastric abscess is by no means an infrequent result of ulcer of the stomach. Lebert observed this complication in 15 per cent. of his own fatal cases and in 12 per cent. of those which he collected from various sources. In our own series of 112 autopsies an abscess was noted in eight cases, and in every instance, with one exception, the gastric disease was of the chronic type. It would also appear that ulceration of the stomach or duodenum is by far the most frequent cause of localised abscess below the diaphragm, since Maydl found it existed in 20 per cent. and Scheurlen in 55 per cent. of the

cases which they collected. Our own experience leads us to believe that nearly 80 per cent. of all cases of subphrenic abscess are dependent upon ulceration of the stomach or duodenum. From our own and other series of cases it would seem that the ratio of perigastric abscess to general peritonitis as the result of perforation is about one to three.

(b) Position and boundaries of the abscess.—An abscess due to gastric ulcer almost invariably occupies the upper part of the left side of the abdomen, lying above and partly in front of the cardiac portion of the stomach, and in immediate contact with the diaphragm. In this position its boundaries are remarkably uniform.

Above, it is limited by the left wing of the diaphragm; below, by the upper surface of the left lobe of the liver, and by adhesions between the anterior wall of the stomach and the

abdominal parietes; the right, by the falciform ligament; on the left, by the spleen, the gastrosplenic omentum, and by adhesions between the cardiac end of the stomach, spleen, and diaphragm; and in front, by abdominal wall and diaphragin. When the abscess cavity is very large, and lies in front of the fundus of the stomach, its lower boundary is usually formed by adhesions between the abdominal wall, colon, and omentum. In only about 6 per cent. of our cases was the absccss situated on the right side of the abdomen. When it occurs in this position

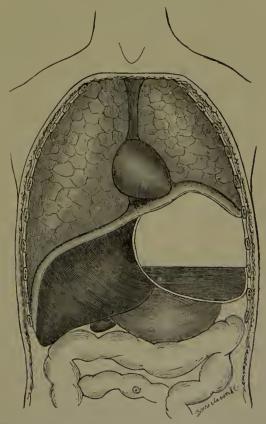


Fig. 25.—Diagram of a perigastric abscess beneath the left wing of the diaphragm.

it is usually bounded above by the right wing of the diaphragm; below, by the right lobe of the liver; on the right, by the side of the thorax; and, on the left, by the falciform ligament. In

rare cases the latter structure is perforated by the pus, so that an abscess forms on either side of it.

Ulcers on the posterior surface of the stomach occasionally perforate into the lesser cavity of the peritoneum, the entrance of which (foramen of Winslow) has been previously closed by adhesions. Under these circumstances an abscess forms which is bounded in front by the stomach; behind, by the pancreas; above, by the liver; and below, by the transverse colon and mesocolon. This variety of perigastric abscess is rarely met with, owing to the firm adhesion which usually exists between the ulcer and the pancreas, but we have seen two examples of it in hospital practice. Chiari has recorded a case in which the pancreas floated as a nccrotic mass in the purulent contents of the cavity, while in one of our cases no trace whatever of this organ could be discovered. An abscess in this situation may either burst into the colon or duodenum, or find its way upwards between the esophagus and the vena cava until it eventually reaches the upper surface of the liver. In both of our cases the patient died while the abscess was still limited to the posterior aspect of the stomach, but Mason has recorded an instance where the pus reached the diaphragm in the manner described, and presented itself on both sides of the chest. Another case is recorded in which the patient died ten days after the commencement of the disease, and where the pus had already found its way to the upper surface of the left lobe of the liver.

Occasionally an ulcer on the upper curvature of the stomach gives rise to a small abscess in the substance of the lesser omentum, while in still rarer instances ulceration near the great curvature is followed by a collection of pus between the layers of the transverse mesocolon.

The contents of a perigastric abscess vary in different cases. When the disease arises from perforation of an ulcer a considerable quantity of gas may escape from the stomach, so that from the first the cavity contains a mixture of air and fluid. In other instances the gaseous element develops at a later date, owing to putrefaction of the contents of the sac. Finally, when the abscess forms without antecedent perforation, its contents may consist solely of pus.

(c) Position of the ulcer.—In the great majority of cases the ulcer which gives rise to a perigastric abscess is situated near

the lesser curvature in the cardiac region. This is due to the fact that it is mainly in this part of the organ that the conditions prevail which favour the formation of a closed cavity. It has already been shown that ulcers near the pylorus usually contract adhesions with the pancreas or liver, while those on the anterior surface give rise to general peritonitis when they perforate the coats of the viscus. But an ulcer near the cardiac end of the lesser curvature, by forming adhesions with the left lobe of the liver, has a cavity ready at hand, into which it can perforate, the only point of danger being the gap between the stomach and the abdominal wall, which, as a matter of fact, becomes rapidly closed by local peritonitis. Into this cavity both the contents of the stomach and the products of inflammation rapidly find their way, being aided by the recumbent position assumed by the patient after the accident, and by the suction action of the diaphragm. This latter factor is of great importance, for it is not only responsible for the tendency exhibited by all abscesses in the abdomen to find their way towards the upper surface of the liver, but is also the chief cause of inflammation of the thoracic organs which so frequently follows subphrenic suppuration.

The following table has been compiled from an analysis of fifty-six cases of perigastric abscess, with the view of showing the usual situation of the gastric ulcer:—

Table 6.—Showing the Situation of the Ulcer in Fifty-six Cases of Perigastric Abscess.

Siti	ation	of ulo	er			No. of cases	Percentage
Pyloric end						12	21.4
Cardiac end	٠	٠	٠	Ŀ.	_:	44	78.5
Anterior surfa						24	43
Posterior surf	ace	÷	·	•		32	57
Near lesser cu					. 1	49	87·5
Near great cu	rvatu	re				7	12.5

Although a large proportion of the cases of perigastric abscess occurs in young women, the ulcer in the stomach is almost invariably of the chronic type, perforation of an acute ulcer being rarely followed by localised suppuration, owing to

the absence of the adhesions which are necessary to prevent general peritonitis.

In about 88 per cent, of our cases a direct communication was found to exist between the stomach and the abscess. most of these perforation had taken place through the base of the ulcer, but in several instances it was expressly stated that either secondary ulceration had occurred in a cicatrix or that adhesions around the base of an ulcer had been ruptured by some violent effort. In the remaining 14 per cent, no perforation of the stomach could be discovered. In some of these it is possible that the aperture may have become obliterated by lymph, and in this way have escaped detection, while in others the ulcer may have healed between the time of perforation and the death of the patient, as in a case recorded by Mason. In the remaining instances, however, it can only be supposed that the abscess was excited without the occurrence of perforation, the septic material being conveyed through the lymphatic channels in the base of the ulcer. That an abscess containing both pus and gas may originate in this manner was well shown in one of our own cases, where two superficial ulcers developed at the cardiac end of the lesser curvature during the course of pyæmia following compound fracture of the forearm. Neither ulcer had penetrated deeper than the muscular coat, but their peritoneal bases were intensely inflamed, and immediately behind the larger one there was an abscess the size of a small egg, containing both gas and offensive pus. In another case a large abscess was associated with an old cicatrix.

When the abscess lies immediately in contact with the stomach, the ulcer opens directly into the sac; but when, as in the case of a pyloric ulcer, the abscess is situated remotely, a fistulous track of considerable length may pass behind the stomach to the left hypochondrium, or upwards to the right lobe of the liver by the side of the gall-bladder.

(d) Complications of perigastric abscess. Inflammation of the thoracic organs.—Secondary inflammation of the thoracic organs, with or without perforation of the diaphragm, occurs in about 80 per cent. of all cases, and usually on the same side as the abscess. These complications are most frequent and severe when the abscess lies in immediate contact with the diaphragm, owing to the ease with which septic material is conveyed by the lymphatics of that structure.

1. Simple dry pleurisy is observed in only about 5 per cent. of all cases. It chiefly affects the base of the lung, which becomes adherent to the diaphragm, and thereby helps to prevent the supervention of more serious results. Inflammation of the pleura with serous exudation was recorded in about 10 per cent. of our cases, but the amount of fluid was usually small

and often encysted by adhesions.

2. Purulent exudation into the pleural cavity occurs in about 12 per cent. of all cases. Occasionally the quantity of pus is so considerable that the disease is mistaken for simple empyema, but more commonly the exudation becomes encysted either at the back of the chest or between the base of the lung and the diaphragm, whence it may find an exit by rupture into a bronchial tube. In some instances the opposite side of the chest is affected in a similar manner.

- 3. Pneumothorax without perforation of the diaphragm may arise from three causes: (1) Putrefaction of the pleural exudation; (2) rupture of a secondary abscess of the lung into the pleura; (3) localised necrosis of the surface of the lung. Leith has related an interesting example of the first condition, and states that two similar cases have been recorded.
- 4. Signs of bronchitis are almost invariably present after death. As a rule this morbid condition is due to cardiac failure during the last stages of the disease, but it may also arise from the coexistence of septicæmia. Pneumonia occurs in about 13 per cent. of the cases, and is usually limited to those parts of the lung which are in immediate contact with the diaphragm. Occasionally the disease affects both sides of the chest. In rare instances, the base of the lung presents a condition of chronic interstitial inflammation. Pulmonary abscess is almost invariably due to pyæmia.
- 5. Pericarditis, without perforation of the diaphragm, is a rare event, and was observed in only 2 per cent. of our cases. It usually arises from inflammation of the left pleura. Endocarditis is never found except as the result of pyemia.
- (e) Modes of termination. Rupture of the sac.—In nearly 60 per cent. of the cases which are not subjected to surgical treatment, the abscess either bursts internally or discharges its contents through the abdominal parietes.
- (1) Perforation of the diaphragm.—This termination was recorded in nearly one-third of our cases, and in about one-half

of those where the abscess burst during life. The rupture of the sac is preceded by local ulceration of the diaphragm, and the channel of communication is usually very small. It is only exceptionally that the aperture is large enough to permit the formation of a diaphragmatic hernia (Günsburg). effects of perforation vary according to the condition of the pleura. If the base of the lung has previously become adherent to the diaphragm, a gangrenous cavity will form in its substance. while, if the union between the two layers of the pleura is incomplete, a localised empyema or pyo-pneumothorax will develop. Finally, if the pleural cavity is in its normal condition at the time of perforation, a diffuse purulent pleurisy or pyo-pneumothorax will result. Perforation of the pericardium is a rare termination of perigastric abscess, and occurred in only about 4 per cent. of our cases. In each instance acute suppurative inflammation ensued.

- (2) Perforation of the colon, &c.—Rupture of an abscess into the transverse colon was observed in 4 per cent. of our cases, while isolated examples of this mode of termination of the disease to the number of about a dozen have been recorded. In most of them the abscess was situated between the stomach and the colon, but in two instances it occupied the lesser cavity of the peritoneum and perforated the posterior wall of the transverse colon (Gairdner). Occasionally the abscess opens into and communicates with both the pleural cavity and the colon (Starcke, Murchison), while perforation of the duodenum or jejunum is also occasionally met with (Fenwick). Rupture of the abscess into the stomach is very exceptional when the disease has originated from simple rulcer.
- (3) Perforation of the abdominal wall was observed only once in our series of cases, but at least six examples of this mode of termination have been recorded by Lloyd, Robertson, Roux, and others. Occasionally the abscess perforates the diaphragm after the latter structure has become adherent to the wall of the chest, and discharges its contents through one of the intercostal spaces.
- (4) Rupture of the abscess into the general cavity of the peritoneum occurred in about 18 per cent. of our cases. In most instances the accident was due to the breaking down of adhesions between the anterior surface of the stomach and the

abdominal wall, especially when the ulcer was situated at some little distance from the lesser curvature (Mason). In rare cases the absects bursts into the subperitoneal tissue, and, passing downwards, points either in the loin or in the iliac fossa, or even finds its way into the pelvis.

Perforation of the duodenum, terminating by the formation of an abscess, is very rare, and has only been observed three

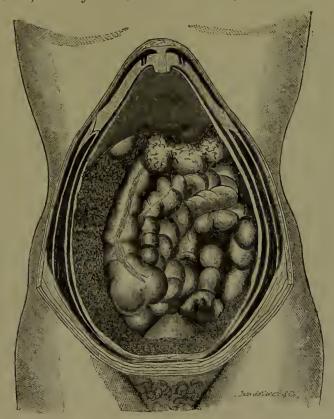


Fig. 26.—Sketch of a case of periduodenal absecss, where the pus found its way behind the peritoneum, and presented in both groins.

times at the London Hospital. We have, however, been able to collect nineteen additional cases from other sources, making in all a total of twenty-two.

In twelve instances the absecss eavity was formed by adhesions between the liver, stomach, intestines, and abdominal wall in the vicinity of the perforation; in one case a fistulous track passed upwards by the side of the gall-bladder to a large collection of pus between the right lobe of the liver and the diaphragm; in another a sinus ran behind the stomach and connected the perforation with an absecss over the left lobe of the liver; in two instances the pus was located in the substance

of the pancreas, while in the remaining six suppuration occurred in the retroperitoneal tissue. In eighteen out of the twenty-two cases the perforation was situated in the first part of the duodenum, and in the other four in the second portion; in each of the latter the abscess was situated behind the peritoneum. In sixteen out of the twenty-two the ulcer occupied the posterior wall of the intestine. Two cases are recorded in which the pus penetrated the cellular tissue of the abdominal wall, and presented externally (Perry and Shaw). Out of the twelve cases of encysted abscess, four burst into the peritoneal cavity, one ruptured into the colon, and two pointed on the right side of the chest (Gross, Streeton). Of the six retroperitoneal cases, five burrowed downwards and presented in one or other iliac fossa, while one appears to have found its way into the posterior mediastinum, and to have pointed in the neck. The usual cause of death was exhaustion, but four died from peritonitis, three from hæmorrhage, two from secondary inflammation of the thoracic organs, and one from pyemia.

7. Perforation of Neighbouring Viscera

A. PANCREAS

In all cases where this gland constitutes the base of an ulcer, the irritation of the gastric juice excites chronic inflammation of its interstitial tissue, which becomes considerably thickened, and the superficial portions of the gland acquire an almost cartilaginous character. With the progress of the disease, however, this adventitious floor becomes gradually dissolved, and a large cavity, filled with ichorous pus, is ultimately formed in the substance of the organ (Goldenberg), into which the ducts may open and allow the secretion of the uninjured portion of the gland to find its way into the cavity of the stomach (Dévic). In other cases the ducts themselves become filled with pus, which finds an imperfect exit into the duodenum. Occasionally the ulcerative process has the effect of completely detaching a portion of the gland, which remains loose in the cavity of the stomach (Rokitanski), while in rare cases the organ is completely perforated, with exposure of the aorta, vena cava, or vertebral column; or the ulcer, after penetrating the pancreas, may establish a communication with the duodenum (Dittrich).

B. LIVER

Like the pancreas, the liver is not infrequently the seat of secondary ulceration. Thus Maygrier records a case where an ulcer on the lesser curvature produced a cavity in the left lobe the size of an orange. When the ulceration takes place rapidly, fatal hæmorrhage often results from the destruction of



Fig. 27.—Chronic ulcer of quadrilateral shape on the posterior wall of stomach, which had formed a deep excavation in the substance of the pancreas. Photograph, much reduced. (London Hospital Museum.)

the hepatic vessels. Perforation of the gall-bladder and biliary ducts is a rare result of gastric ulcer, although cases of this nature have been recorded by Rokitanski, Dittrich, Barker, Habershon, Long, and others.

C. SPLEEN

Owing to its remote situation from the usual site of a gastric ulcer, the spleen is seldom involved by the disease. Fatal hamorrhage, however, has occasionally ensued from erosion of its pulp.

D. PERFORATION OF THE DIAPHRAGM. GASTRO-THORACIC FISTULÆ

Direct perforation of the diaphragm by a gastric ulcer is very rare, and has only once been recorded at the London Hospital during a period of forty years; while after a careful search through the literature we have only been able to find

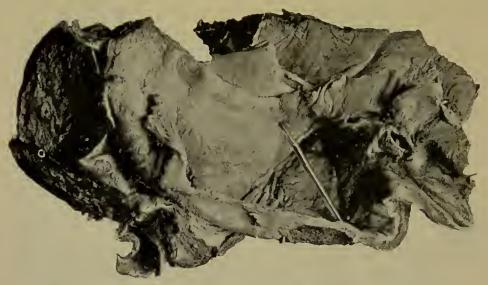


Fig. 28.—Photograph of the inner surface of the stomach. Above and to the right of the glass rod is a chronic ulcer, which penetrated through the diaphragm into the left pleural cavity. (London Hospital Museum.)

twenty authentic cases. In every instance the perforation was brought about by the gradual erosion of the muscle by a chronic ulcer which had become adherent to it.

Direction of the Perforation.—It has already been shown that when a perigastric abscess bursts into the thorax it almost invariably finds its way into the left pleural cavity, while rupture into the pericardium only occurs in about 4 per cent. of the cases. When, however, the diaphragm is directly perforated by an ulcer, it would appear that the heart is almost as frequently affected as the lung, since no less than nine out of our twenty-one cases (43 per cent.) involved the pericardium.

Biach (Wien. Med. Wochen. January 30, 1880) found that out of 918 cases of pyo-pneumothorax, only two were due to the perforation of a gastric ulcer.

This notable contrast between the two modes of perforation is mainly due to the different location of the two diseases. Whatever be the position of the ulcer, a perigastric abscess almost invariably forms beneath the left wing of the diaphragm, and is consequently more likely to burst through the contiguous portion than to seek a more remote and difficult exit through the central tendon. A chronic ulcer, on the other hand, can only become adherent to that part of the diaphragm with which it lies in contact; and since the disease is much more common on the lesser curvature than in the fundus of the stomach, it follows that the pericardial portion of the structure is quite as likely to be perforated as the pleural. In rare cases the ulcer penetrates the posterior part of the diaphragm near its centre, and thus opens up a direct communication between the stomach and the mediastinum.

Position of the Ulcer.—In view of the anatomical relations of the stomach and diaphragm it might be surmised that perforation of the chest can only occur when the ulcer is situated either in the fundus of the organ or near the lesser curvature in the vicinity of the cardiac orifice. This supposition is confirmed by the fact that, in every case which has hitherto been recorded, the ulcer was found to occupy one or other of these positions.

Table 7.—Showing the Results of Direct Perforation of the Diaphragm in Twenty-one Cases of Gastric Ulcer

Position of ulcer			Number of cases	Left pleura	Peri- cardium	Heart	stinum
Fundus	•		6 15	5 6	1 4		<u> </u>
Total	•	•	21	11	5	4	1

Results of Perforation.—(a) Left pleura.—The immediate effect of perforation of the left side of the chest varies with the condition of the pleura at the time of the accident. If the local inflammation which accompanies ulceration of the diaphragm has not been sufficient to produce adhesion between the muscle and the base of the lung, the rupture will allow the contents of the stomach to enter the serous sac. If the stomach contains a large quantity of food, the irritation of

the chyme will excite acute pleurisy, usually of a suppurative type; while, if the contents of the stomach consists principally of gas, the initial phenomena will be those of pneumothorax.

Thus, out of our eleven cases of perforation of the left pleura, empyema was noted in three instances (Siebert, Greiger, Günsburg), 'exudation' in one (Sicherer), pneumothorax in one (Müller), and pyo-pneumothorax in three (Kogerer, Starcke, Authors). In two of the cases (Sicherer, Müller), one or more round worms were also found in the serous cavity. In rare instances the hole in the diaphragm is so large that the stomach or spleen may enter the thorax (diaphragmatic hernia: Günsburg, Needon). On the other hand, if the antecedent pleurisy has produced adhesion between the base of the lung and the diaphragm, the perforating ulcer will make its way directly into the pulmonary tissue, and produce a gangrenous cavity which may finally establish a communication between the stomach and a bronchial tube. This condition was observed in three out of the eleven cases (Rokitanski, Günsburg, Aufrecht).

- (b) Pericardium.—The results of perforation of the pericardium are essentially the same as those just described. In two out of the nine cases pneumopericardium was found after death (Säxinger, Fenwick), while in two others the sac contained both pus and air (Cérenville, Guttmann), and in one fluid and air (Rosenstein). In four cases the cavity of the pericardium had been either partially or completely obliterated by antecedent inflammation, so that the ulcer was enabled to penetrate the muscular tissue of the heart (Oser and Chiari, Brenner, Finny, Bruenniche). In each instance the posterior wall of the left ventricle near the apex was the site of the perforation, and death ensued from hæmorrhage into the stomach and intestines.
- (c) Mediastinum.—The only authentic case of perforation of the mediastinum is recorded by Faber, in which an ulcer on the lesser curvature perforated the centre of the diaphragm during an attack of retching. The posterior mediastinum became distended with gas, which finally penetrated the anterior mediastinum and found its way thence into the subcutaneous tissues of the body.¹

¹ The cases by Korach and Pönsgen are incorrectly cited as examples of perforation of the mediastinum.

E. PERFORATION OF THE BOWEL. INTERNAL GASTRIC FISTULÆ

(1) Gastro-colic Fistula.—Direct perforation of the transverse colon by an ulcer of the stomach was first described by Haller in 1744. That the condition is a rare one is shown by the fact that in 1857 Murchison was only able to find eight recorded examples, while during the last forty years only two cases have been observed in the post-mortem room of the

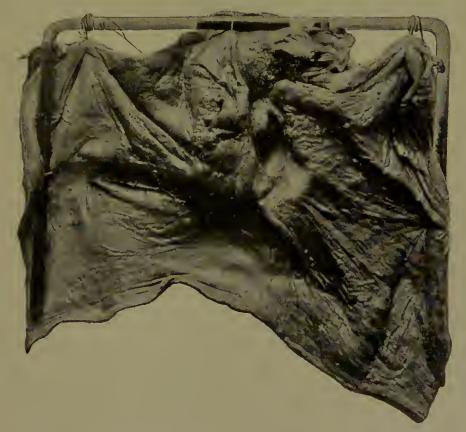


Fig. 29.—Photograph of the inner surface of the transverse colon, showing a fistulous opening produced by a simple ulcer of the stomach. Natural size. (London Hospital Museum.)

London Hospital. In 75 per cent. of the cases which have been published up to the present time, the ulcer was situated close to the great curvature of the stomach, and in the remaining 25 per cent. either at the pylorus or in the fundus of the organ. In every instance the ulcer had first contracted

¹ Murchison mentions 'nine or ten cases' in his monograph, but two appear to have been doubtful.

adhesions with the bowel, and afterwards gradually perforated its wall. As a rule the opening in the intestine is very small, but in at least two of the recorded cases (Levenstein, May) it exceeded an inch in diameter. In several instances the intestinal orifice was divided into two by a slip of mucous membrane, while prolapse of the inner coat of the colon was found, in a few cases, to constitute a kind of valve. It is interesting to note that this variety of fistula is twice as common in cancer of the stomach as in simple ulcer, owing probably to the greater frequency of malignant disease in the vicinity of the great curvature, and to its tendency to involve the neighbouring viscera.

(2) Bigastric Fistulæ.—It occasionally happens that an ulcer situated at the cardiac end of the lesser curvature contracts adhesions to the upper and posterior aspect of the pylorus, with the result that the viscus becomes twisted or bent upon itself. Should the ulcer perforate, a fistulous communication is established between the two portions of the stomach.

(3) Gastro-duodenal Fistulæ.—Gastro-duodenal fistulæ almost invariably arise from the perforation of an ulcer into the third portion of the duodenum. In some of the recorded cases the ulcer was situated on the posterior wall of the stomach immediately below the pylorus, so that when a fistula was established the pyloric ring formed a kind of bridge over the gastric opening (Rokitanski, Dittrich). In rare instances, an ulcer on the lesser curvature becomes adherent to the anterior or upper surface of the first part of the duodenum, and, eventually penetrating the wall of the bowel, permits the food to pass directly into the gut. In such cases the pylorus usually becomes contracted (Thierfelder). In an interesting case recorded by Mohr, an ulcer on the posterior surface of the stomach produced a large cavity in the head of the pancreas, which eventually opened into the lower part of the duodenum. In this manner a communication was established between the two hollow viscera through the substance of the gland. A direct communication between the stomach and other parts of the small bowel has never been recorded.

A duodenal ulcer may establish a fistulous communication

¹ The case recorded by Jones (*Trans. Med. Chir. Soc.* vol. xxxv. p. 35), of gastrocolic fistula in a boy five years of age, was probably one of congenital dilatation of the colon, with secondary ulceration.

with the gall-bladder, colon, or stomach. In rare cases it perforates the wall of the chest or the abdominal parietes (Perry and Shaw).

F. PERFORATION OF THE SKIN. EXTERNAL GASTRIC FISTULÆ

An ulcer of the stomach may lead to the formation of a gastro-cutaneous fistula, either directly by adhesion to and perforation of the abdominal parietes, or indirectly through the medium of a localised abscess. Out of the twenty-eight cases of gastro-cutaneous fistula collected by Murchison, six were due to cancer and twelve to simple ulcer. In three of those arising from ulcer the disease appeared to have originated in the direct manner, and in five from the bursting of a perigastric abscess (indirect). In the former cases the actual perforation of the skin was preceded by the formation of a hard, tender turnour, which finally ulcerated through the parietes after an interval of some months or even years, while in the latter the turnour was less circumscribed but more rapid in its development, and was accompanied by the usual signs of subcutaneous abscess.

The situation of the external opening varies with the location of the ulcer. Thus, when it occurs at the pylorus, the fistula usually opens near the umbilicus, while if it is situated in the centre of the organ or in the fundus, the external orifice occupies the left hypochondrium or epigastric region. In very rare cases the fistula traverses the diaphragm and opens on the surface of the chest between the ribs. The first recorded instance of a gastro-thoracic-cutaneous fistula which we have been able to find was published by Wencher in 1743, and since that date only five others have been described (Middledorpf, Grünewaldt and Schröder, König, Rokitanski, Fillenbaum). There is also one where the ulcer was situated in the first part of the duodenum (Streeton). With one exception (Wencher), the perforation of the chest wall was always brought about in the direct manner. An interesting case has been recently published by Pick, in which an ulcer close to the pylorus perforated the diaphragm and gave rise to a hard inflammatory mass in the left thoracic wall, which was diagnosed as a sarcoma. The skin of the chest eventually became emphysematous, but the patient died of tuberculosis before a fistula was actually established.

The edges of the external orifice are round, hard, and somewhat depressed, so that the aperture presents the appearance of a funnel. The surrounding skin is usually reddened, thickened, and excoriated owing to the constant irritation produced by the acid contents of the stomach.

8. Diseases of the Stomach Secondary to Ulceration

- (1) Chronic Gastritis.—Every chronic ulcer of the stomach is accompanied by some degree of gastric catarrh. In many cases the inflammatory affection is limited to the immediate neighbourhood of the sore, and is responsible for the thickened and sclerosed appearance of the tissues around the ulcer. When the pyloric orifice is obstructed the whole of the stomach is apt to become affected with a form of chronic parenchymatous and interstitial inflammation, accompanied by hypersecretion of the gastric juice (Korczinski and Jaworski).
- (2) Phlegmonous Gastritis of a circumscribed character occasionally arises from simple ulcer. In two of our cases a small abscess was observed in the wall of the stomach within a few inches of the ulcer. In each instance the cavity was situated in the submucous coat, and was covered by a thin layer of mucous membrane, while the deeper tissues were softened, infiltrated, or partially destroyed. In one the abscess was about the size of a walnut, and contained more than a drachm of pus, while in the other the sac had discharged its contents during life through several small orifices in the mucous membrane. The only case of diffuse phlegmonous gastritis with which we are acquainted is the one recorded by Hemmeter, where the disease followed cancerous infiltration of a simple ulcer.
- (3) Localised Cirrhosis of the Stomach, due to chronic inflammation, is often observed around the edges of an ulcer. In two of our cases the whole of the pyloric region of the organ presented a leather-bottle appearance from this cause, and others have been recorded where a similar thickening of the gastric tissues led to an erroneous diagnosis of cancer.

75

Diffuse cirrhosis is extremely rare. Leith, however, has recorded a case in which the cirrhotic process appeared to have originated in the scars of some ulcers from which the patient had suffered ten years previously. The whole of the organ was much thickened and contracted, and the pylorus

partially stenosed.

(4) Cancer.—The invasion of an ulcer or its scar by carcinoma was first observed by Cruveilhier, and afterwards investigated more fully by Rokitanski, Brinton, and Dittrich. According to Lebert, about 9 per cent. of all gastric cancers originate in a simple ulcer; but Rosenheim is inclined to reduce this estimate to 6 per cent., and Haberlin to 2-3 per cent. Zenker, on the other hand, believes that most cancers of the stomach are preceded by an ulcer. Only two cases of this description have come under our personal observation, and we have only been able to find three instances recorded in the Transactions of the Pathological Society of London. Our first case occurred in a man who had suffered from hæmatemesis for many years, and who finally died with a large scirrhous tumour involving one side of a chronic ulcer. In the second an elderly man died from scirrhus en cuirass, and at the autopsy two tumours were found in the edge of a large chronic ulcer whose presence had been unsuspected during life. Microscopical examination proved that the tumours consisted of scirrhous cancer. As a rule the malignant disease is quite circumscribed, and seldom involves the surrounding mucous membrane. It usually exhibits the characters of scirrhus, but instances of encephaloid cancer have been recorded. Hauser's investigations concerning the healing of ulcers and the development of adenomata in the scar tissue have already been noticed (p. 38).

Duodenal ulcer is sometimes followed by carcinoma. Perry and Shaw give the notes of five cases of this description, but we have not found any record of its occurrence in the postmortem books of the London Hospital.

9. Diseases of other Organs associated with Ulcer of the Stomach

It is important to observe that in nearly 75 per cent. of the cases where an open ulcer has been found in the stomach after death, one or more important organs of the body have also been affected with organic disease. According to Papellier, tubercle of the lung is encountered in 18 per cent. of all cases of gastric ulcer, while Engel and Jaksch estimated its frequency at 19 and 20 per cent. respectively. These statements appear to be substantially correct, since we find that in 993 autopsies upon gastric ulcer, 169, or 17 per cent., presented evidence of tubercular disease of the lungs. In most cases the pulmonary complaint was very chronic in character, and only in about 3 per cent. of the cases was the process described as 'recent' or 'acute.' Among the other diseases to which the lungs are liable, acute pneumonia was noted in 13 per cent. of our collected cases, thickening or adhesions of the pleura in 6 per cent., and emphysema in 4 per cent.

Chronic endocarditis, principally affecting the mitral valve, was present in 9 per cent., but acute endocarditis was only observed in the pyemic cases. Other lesions of the circulatory organs, such as enlargement of the heart, atheroma of the great vessels, aneurism and thrombosis were present in 19 per cent. of the cases.

In 8 per cent. the kidneys were affected with chronic interstitial inflammation, while in nearly 9 per cent. the liver showed signs of alcoholic cirrhosis. Gummata, cicatrices, or other evidences of syphilis, were noted in 6 per cent.; and in 3 per cent. cancer and simple ulcer were present in the same stomach.

It has been stated that pyamia may arise from absorption through the base of a gastric ulcer in the same way that a wound of the surface of the body may prove the source of a general infection. That pyamia does occasionally ensue from ulceration of the stomach is beyond question, but there is no evidence to show that it originates in the manner indicated. On the contrary, in every case with which we are acquainted, the systemic infection was preceded either by a localised abscess in the peritoneum or by a septic cavity in the pancreas, liver, or spleen. Under such conditions it is usual to find multiple abscesses in the liver and lungs, with secondary inflammation of the pleura, pericardium, or endocardium (Murchison, Leith). It must be remembered, however, that not only is gastric ulcer itself a frequent result of general pyamia, but that the two diseases may co-exist without there

being any causal relationship between them. It is due to ignorance or forgetfulness of these elementary facts that many diseases which occur accidentally in the subjects of ulcer of the stomach are attributed to the influence of the gastric complaint.

CHAPTER III

THE ETIOLOGY OF GASTRIC AND DUODENAL ULCER

1. Frequency

The average frequency of gastric ulcer may be determined in two ways, both of which, however, are open to serious objections. The usual plan adopted is to note the number of open ulcers and scars which occur in the stomach in a large series of autopsies performed on persons dying from different diseases. From calculations made upon this basis Brinton stated that ulceration of the stomach occurs in about 5 per cent. of the total number of deaths from all causes. Out of 47,912 autopsies we have collected from different sources either an ulcer or cicatrix was present in the stomach in 2,019, or in about 4.2 per cent., a result which tallies closely with the conclusions arrived at both by Lebert and Welch. It would therefore seem that about 4 to 5 per cent. of the population suffer at one time or another from this disease.

Owing to the ease with which small scars are overlooked, and the liability of mistaking cirrhotic patches in the mucous membrane, due to chronic catarrh, for evidences of former ulceration, it is obvious that computations based upon the frequency of scars in the stomach can only possess a comparative value. A more important point is to determine the average frequency of an open ulcer. For this purpose we have examined the records of 20,317 autopsies, and find that a gastric ulcer was present in 281 cases, or in about 1.37 per cent. It is probable, therefore, that the disease exists in a more or less active state in about 1.5 per cent. of persons dying from all causes.

Although our knowledge of the subject is at present very defective, the disease appears to be more common in Northern than in Southern Europe, and to be less frequent in France than in most of the neighbouring countries. The average frequency of gastric ulcer in some of the large cities of Europe, as

determined by post-mortem statistics, is as follows: Berlin, 3.6 per cent.; Copenhagen, 13 per cent.; Dresden, 11 per cent.; Dublin, 10 per cent.; Erlangen, 4.5 per cent.; Jena, 10 per cent.; Kiel, 8.3 per cent.; London, 4.6 per cent.; Munich, 1.23 per cent.; Prague, 4.5 per cent.; Zurich, 2.16 per cent. According to Da Costa and Welch, it is less prevalent in America than in Europe. Sperk states that gastric ulcer is very common in Eastern Siberia, while Sohlern found it was comparatively rare over the greater part of Russia. Palgrave is cited by Welch as having noted its frequent occurrence in Arabia.

Clinical experience appears to indicate that gastric ulcer is much less common than pathological evidence would lead us to believe. Thus Lebert only diagnosed the disease in 0.6 per cent. of the 41,688 cases which came under his care in Zurich and Breslau; while out of 45,712 cases admitted into the London Hospital and London Temperance Hospital in the course of ten years, only 383, or about 0.82 per cent., were supposed to be suffering from gastric ulceration. These figures agree very closely with those compiled from outpatient practice, since we find that out of 5,000 consecutive cases that attended the last-named hospital between June 1897 and January 1899, only thirty-seven, or 0.74 per cent., presented the clinical features of the complaint.

The duodenum is much less frequently affected with simple ulcer than the stomach. Perry and Shaw state that the disease occurred in 0·4 per cent. of the cases which were examined at Guy's Hospital between the years 1826 and 1892; while in 13,055 autopsies, where the condition of the small bowel was noted, we find that an open ulcer of the duodenum was present in 34, or in 0·26 per cent. Bearing in mind the comparative infrequency with which an ulcer in this region heals, it appears probable that chronic ulcer of the stomach is about ten times as common as ulcer of the duodenum. According to our statistics, only about 1·7 per cent. of the cases of gastric ulcer are accompanied by a similar condition of the duodenum.

The statistics from Berlin (14,537 autopsies) were compiled by Plange, Steiner, Wollmann, and Berthold; from Copenhagen (200), by Dahlerup; from Dresden (2,878), by Stachelhausen; from Dublin (250), by McWeeney; from Erlangen (1,166), by Ziemssen; from Jena (384), by Starcke; from Kiel (1,658), by Greiss; from London (10,000), by Fenwick; from Munich (3,500), by Nolte; from Prague (11,888), by Jaksch, Dittrich, Willigk, and Eppinger; from Zurich (3,476), by Stoll.

2. Sex

It is generally admitted that ulcer of the stomach is more common in women than in men. Out of 2,031 cases of open ulcer which we have collected from various sources 1,227 occurred in females and 804 in males, or in the proportion of 3 to 2. Clinical experience, however, indicates that the liability of women to the disease is far greater than the above figures appear to show. Thus, of 209 cases which Lebert diagnosed as gastric ulcer, 159 were women and only forty-seven men, or in the proportion of $3\frac{1}{2}$ to 1. In our own series of 383 cases, the great majority of whom had suffered from hæmatemesis, 288 were women and ninety-five men, a ratio of 3 to 1. The only conclusion which can be arrived at by a comparison of these two sets of figures is that, while women are far more subject to the complaint than men, they are also less liable to die from it.

It has already been stated that acute ulcers heal rapidly, and, except when perforation occurs, seldom terminate fatally; while the chronic form of the disease usually exhibits a progressive character, and heals slowly and often imperfectly. It is therefore probable that the divergence between pathological and clinical experience as to the frequency of the disease in the two sexes may be explained by a greater liability on the part of women to the acute complaint. Unfortunately the various hospital statistics, from which most of our material has been derived, fail to differentiate between the two forms of the disease, so that we are obliged to have recourse to our own cases, which are analysed in the following table:—

TABLE 8.—EIGHTY-NINE CASES OF OPEN ULCER, ARRANGED ACCORDING TO THE CHARACTER OF THE DISEASE AND AGE OF THE PATIENT AT THE TIME OF DEATH

Age	Acut	e ulcers	Chronic ulcers		
WRG	Males	Females	Males	Females	
10-20 20-30 30-40 40-50 50-60 60-70 70-80	1 1 1	7 13 7 — —	$-\frac{1}{5}$ $\frac{5}{22}$ $\frac{8}{6}$ $\frac{1}{1}$	1 2 3 6 4	
Total	3	27	43	16	

It will be seen that whereas acute ulceration of the stomach is almost entirely confined to young females, the chronic form of the disease is much more common in the male sex.

Duodenum. - All writers are agreed that simple ulcer of the duodenum is far more common in men than in women. Krauss estimated the ratio at 10 to 1, Trier at 5 to 1, Cullen at 5\frac{1}{2} to 1, and Perry and Shaw at 3 to 1. In our own series of cases, the proportion of males to females was nearly 4 to 1. When, however, we exclude those ulcers which were secondary to burns, pyæmia, cardiac or Bright's disease, and divide the remainder into 'acute' and 'chronic' according to the appearance of the lesion, we find that in the acute idiopathic variety the ratio of males to females was 5 to 1, and in the chronic form 10 to 1. It is obvious, therefore, that Krauss must have collected only examples of the ordinary chronic disease, while the other authors included both varieties in their respective series. This conclusion is borne out by an examination of the different cases which are recorded in the paper by Perry and Shaw.

In the case of duodenal ulceration secondary to burns, the relative liability of the sexes is reversed, females being nearly twice as often affected as males. This phenomenon may be due to the fact that women are exceptionally prone to suffer from severe burns on account of their loose clothing.

3. Age

The period of life at which the disease is most apt to develop has been the subject of numerous investigations. Brinton attempted to settle the question by collecting a number of autopsies in which evidence of ulceration of the stomach had been present; but, owing to the fact that his statistics included both scars and open ulcers, he came to the erroneous conclusion that the liability to the disease increases with the progress of life. The cause of this mistake is well shown in the following table, where an equal number of open ulcers and scars are arranged according to the age of the patient at the time of death. It will be seen that, whereas open ulcers diminish in frequency after the age of fifty, the

cicatrices, being ineffaceable records of the disease, apparently increase in number with each decade.

TABLE 9.—REPRESENTING SIX HUNDRED CASES OF ULCER OR SCAR IN WHICH THE AGE IS GIVEN AT THE TIME OF DEATH

Age	Open ulcers (300 cases)	Scars (300 cases)	Totals (600 cases)
10-20	21	8	29
20-30	65	49	114
30-40	48	50	98
40-50	62	40	102
50-60	48	53	101
60-70	39	65	104
70-80	12	24	36
80-90	5	9	14
90-100		2	2

In order to avoid this error we have collected 825 cases where an open ulcer was discovered after death (Table 10). It will be seen that the disease is most often encountered on the post-mortem table between the ages of twenty and thirty, while from thirty to sixty the cases are distributed nearly equally over the three decades.

Table 10.—Showing the Age at Time of Death in 825 Cases of Gastric Ulcer

Age	No. of Cases	Age	No. of Cases
10-20 20-30 30 40 40-50 50-60	47 167 139 147 144	60-70 70-80 80-90 90-100	123 47 10 1

These statistics, however, afford no clue as to the age at which the complaint commenced in the various cases; they merely indicate the relative frequency of open ulcers in the stomach at different periods of life. In order to determine the all-important question as to the age at which the complaint usually develops, we have analysed our 383 clinical cases according to the age of the patient when the first symptoms manifested themselves.

It will be observed that there is a remarkable difference in the age-incidence of the disease in the two sexes. In males, more than one-half of the cases occur between thirty and

TABLE 11.—ANALYSIS OF 383	Cases of Gastric Ulcer, showing the Age at	
WHICH	THE DISEASE COMMENCED	

Age	Males	Females
10-20 20-30 30-40 40-50 50-60 60-70	$egin{array}{c} 7 \\ 22 \\ 33 \\ 23 \\ 6 \\ 4 \\ \end{array} egin{array}{c} 30\% \\ 59\% \\ 6 \\ 4 \\ \end{array}$	$egin{array}{c} 68 \ 147 \ 147 \ 13 \ 13 \ 0 \ \end{array} egin{array}{c} 75\% \ 49 \ 13 \ 0 \ \end{array}$
Total	95	288

fifty years of age, while in females three-quarters of the entire number commence before the age of thirty. The cause of this phenomenon is to be found in the fact already noticed, namely, that young women are unduly liable to acute ulceration of the stomach, while males of middle age are most prone to suffer from the chronic form of the disease (Table 8).

Death from ulcer of the stomach is rare after the age of seventy, although Weber has recorded a case of fatal hæmatemesis in a woman aged seventy-two, and Sedgwick perforation in a man eighty-two years of age. A patient who was under our observation died from it when over eighty years of age, whilst Eppinger mentions the case of a man whose age was reputed to be 120 years. Simple ulcer of the stomach is very rare before puberty. Welch only found one instance of the disease in the first decade of life among the 607 cases which he collected, while Rokitanski, in the whole of his wide experience, never encountered it in a child less than fourteen years of age. The records of the London Hospital contain three cases of ulceration of the stomach in young children, but in each instance it was secondary to some other complaint. After a careful search through the literature we have only been able to find eighteen genuine examples of ulcer of the stomach in infancy and childhood, of which thirteen belonged to the acute and five to the chronic form of the complaint. It is interesting to observe that the proportion of females to males was 4 to 1.1

¹ For literature see Author's Diseases of Digestion in Infancy and Childhood, p. 287.

Duodenum.—Ulcer of the duodenum is said to be most frequent about the age of thirty-eight. This, however, is merely the average age at which death occurred in a large series of unclassified cases. When a proper distinction is made between the acute and chronic forms of the disease, with the exclusion of those which have arisen from pyæmia and other constitutional or organic affections, the results closely approximate to those which have already been obtained in the case of ulcer of the stomach.

Table 12.—Showing the Age at the Time of Death in Sixty-eight Cases of Duodenal Ulcer

Age	Acute ulcers	Chronic ulcers
15-20	4	
20-30	13	6
30-40	3	12
40-50	2	15
50-60	2	7
60-70	1	3
Total	25	43

It will be seen that, whereas the vast majority of the acute cases (68 per cent.) occur between the ages of fifteen and thirty, chronic ulcer is most frequent (63 per cent.) between thirty and fifty years of age.

4. Occupation

The form of occupation does not appear to exercise any decided influence upon the development of the disease. The various professions which are attended with special anxiety and mental strain, while they are extremely apt to engender functional disorders of the digestive system, do not predispose to the inception of gastric ulcer; nor as a rule do those who live by manual employments suffer especially from it. Eichorst states that ulceration of the stomach is very common among metal turners, owing to irritation of the stomach produced by the particles of metal which are constantly swallowed, and Bernutz believes that porcelain makers are also liable to the disease for a similar reason. We have seen several cases of chronic ulceration of the stomach among knife-grinders,

metal turners, and other persons engaged in dusty occupations, but inquiries made among their fellow-workinen always failed to establish the fact that the complaint was exceptionally rife among them. Tailors, weavers, and shoemakers were considered by Habershon to be unduly susceptible to the disease, owing to the pressure exercised upon the pit of the stomach during the performance of their work, while others have supposed that the use of tight belts or corsets produces a like prejudicial effect by causing partial anæmia of the gastric tissues. These and other statements of a similar character are unsupported by any material evidence.

The well-known fact that maidservants, dressmakers, and governesses are very prone to suffer from hæmatemesis and perforation of the stomach has led to the belief that occupations of this nature are directly productive of gastric ulcer; indeed, Bamberger went so far as to state that the frequent occurrence of the complaint in young cooks was due to their habit of constantly tasting hot foods. But apart from the fact that cooks are not more liable to the disease than other domestic servants, it is clear that all the persons who follow these several employments are women who have arrived at the very age when acute gastric ulcer is most common, so that it is quite admissible to assume that the age and sex of the individual are more important from an etiological point of view than the occupation. This supposition is confirmed by the fact that the disease is very rare among young men who follow sedentary employments, such as footmen, clerks, and telegraphists. It must therefore be concluded that indoor occupations, by inducing a state of general ill-health or anemia, merely increase the natural predisposition of young women to acute ulcer of the stomach.

5. Hygiene and Habits

The disease is more common among the poor than among the rich, and more often attacks the inhabitants of large cities than those who live in rural districts. These facts have led to the assumption that insufficient nourishment, bad ventilation, and exposure to cold and wet are important factors in the etiology of the complaint. It does not appear, however, from the inquiries which we have made in various workhouse infirmaries, that ulcer of the stomach is exceptionally frequent

among the waifs and strays of London, who are pre-eminently exposed to these different conditions, while the mere fact that it attacks all classes of society demonstrates that defective hygiene is not essential to its development.

Certain writers have endeavoured to prove that the greater liability of the poor to the disease arises from an excess of vegetables in their diet. But even if it be allowed that the labouring classes subsist chiefly on vegetables—a supposition which is totally at variance with fact—such a theory hardly harmonises with the researches of Sohlern, who found that gastric ulcer is practically unknown among the inhabitants of Greater Russia, the Bavarian Alps, and certain parts of Germany, who live almost entirely upon vegetables, nor yet with the circumstance that the complaint is less common among agricultural labourers than among the workers in great cities. Domestic servants, again, who are unusually susceptible to the disease, are notoriously great eaters of meat; indeed, in several cases which have come under our notice, it seemed as if the incidence of the disease was determined by a sudden change from a meagre diet to the gross feeding of the servants' hall.

According to Lanceraux, the abuse of alcohol, especially in the form of ardent spirits, is a frequent cause of the complaint. So far as the chronic form of the disease is concerned this statement is probably correct, as we find that 14 per cent. of our hospital cases were admittedly hard drinkers, while in 9 per cent. of the fatal cases the liver presented signs of cirrhosis. But with regard to the acute form of the complaint there is no evidence to show that it has any connection with the use of alcoholic drinks; for not only is it quite as common among teetotallers as among those who take stimulants, but the mere fact that it occurs almost exclusively in young women is sufficient to eliminate alcohol as a factor in its production.

6. Heredity

There does not appear to be any particular inherited predisposition to the disease. In about 3 per cent. of our cases one of the parents was said to have suffered from the complaint, and in nearly 5 per cent. some other member of the

Nearly 85 per cent. of the cases admitted into the London Temperance Hospital were total abstainers.

family had presented similar symptoms. In almost every instance, however, the disease was of the acute type and affected the young female members of the family. The occurrence of chronic ulcer of the stomach in successive generations is extremely rare.

7. Traumatism

Severe blows upon the epigastrium are not infrequently followed by hæmatemesis and other symptoms of gastric ulcer which may persist for several weeks or months; but superficial injuries inflicted upon the inner surface of the stomach by swords or other sharp instruments which have been swallowed usually heal without creating any local disturbance. The reason why an external injury is so apt to be followed by ulceration may be explained by the hæmorrhagic infiltration of the coats of the organ that takes place in these cases, which leads to a localised necrosis of the mucous membrane that requires a considerable time for its proper repair.

The ingestion of corrosive poisons is another variety of direct injury to the stomach which is apt to be followed by an enduring type of ulceration, for in such cases it is not unusual to find a chronic sore in the neighbourhood of the pylorus long after the superficial injuries caused by the fluid have disappeared (Fox, Williams, Fenwick). This peculiar location of the ulcer is due partly to the fact that the corrosive substance, being unable to escape into the duodenum owing to the spasmodic closure of the pyloric orifice, exerts a more lasting effect upon the tissues of this region, and partly to certain anatomical peculiarities of the structures which retard the processes of repair. Superficial burns are followed by acute ulceration of the duodenum in about 6.2 per cent. of all cases, and occasionally by a similar lesion of the stomach. (Holmes, Erichsen, Perry and Shaw.) Frost-bite is also stated to give rise to the disease.

8. Excessive Secretion

Since Reichmann in 1882 drew attention to the fact that hypersecretion of the gastric juice frequently accompanies ulceration of the stomach, the idea that the organic lesion owes its origin to the functional disorder has almost become an article of faith with the majority of Continental physicians.

According to this theory the stomach does not digest itself under ordinary circumstances because the acid secretion is only called forth by the introduction of food, and ceases as soon as its work is completed; but when it occurs in the intervals of digestion it erodes the wall of the organ for want of some other material upon which to act.

Most writers of the present day are agreed that chronic ulcer of the stomach is often accompanied by an excessive acidity of the gastric juice. Riegel found an excess of hydrochloric acid in every one of the thirty-one cases which he examined, while Schaeffer, Korczinski and Jaworski, Bouveret, and many others have attested the constant association of the two complaints. On the other hand, Ritter and Hirsch, Müller, Lenhartz, Cahn and v. Mering, Gerhardt and Ewald have recorded instances in which the acid was either normal or diminished in quantity.

In this country the stomach-tube has never attained any degree of popularity as a means of diagnosis, partly because the majority of patients have a decided objection to its employment, and partly because English physicians have always held that an ulcerated stomach should be treated with the greatest care, and that even the application of external pressure is not devoid of danger. For our own part, we have known so many accidents ensue from exploration of the stomach that we never permit a tube to be employed in a case of gastric ulcer unless it be to relieve the effects of pyloric stenosis, and we are consequently unable to offer any opinion as to the usual percentage acidity after a test meal. We find, however, that nearly 72 per cent. of our cases who vomited after meals presented an excess of hydrochloric acid in the ejecta, a result which closely agrees with Rosenheim's estimate of the frequency of hyperacidity in gastric ulcer, viz. 66 per cent.

The theory which attributes an ulcer of the stomach to auto-digestion is very fascinating, but to the clinician it presents certain difficulties of acceptance. In the first place, if the gastric juice is free to act upon any part of the gastric wall, why does it habitually select the most inaccessible part of the organ, viz. the upper margin, to the exclusion of the fundus which is constantly bathed in the destructive fluid, and which invariably suffers most from post-mortem digestion? and why does it only produce a solitary ulcer of comparatively small

size instead of the diffuse softening with which pathologists are so familiar? Again, if hyperacidity is a necessary factor in the production of an ulcer, why does it not exist in cases of the acute perforating disease in young women? It is true that Grüne is stated to have observed an excess of hydrochloric acid in all the cases he investigated; but his observations are diametrically opposed to those of Müller, while in nineteen cases of acute ulcer, in which we examined the contents of the stomach a month after an attack of hæmatemesis, not only was the total acidity of the filtrate invariably diminished, but only in two instances could any trace of free hydrochloric acid be detected. Of course it is open to argument that the hyperacidity may have disappeared after hæmorrhage had occurred; but against this supposition is the fact that we have never been able to determine the presence of an excess of acid in young women suffering from the dyspepsia of chlorosis. Lastly, is it not surprising that a lesion which is supposed to owe its origin to the abnormal condition of the gastric secretion should so often heal in spite of the continued existence of its exciting cause? That this does occur is shown by the frequent discovery of cicatrices in the pyloric region after death from hypersecretion; indeed, we have never witnessed an autopsy upon a case of Reichmann's disease where there was not either a scar or some other cause of pyloric stenosis. For our own part, we believe that the relationship between hypersecretion and gastric ulcer is somewhat as follows.

An ulcer commences as a localised necrosis of the mucous membrane, which subsequently undergoes digestion or exfoliation. As a rule the loss of substance rapidly heals, but if any local or constitutional condition is present which hinders the process of repair, the ulcer, instead of healing, may assume a progressive character. Among the various causes which prevent repair an excessive acidity of the gastric juice is obviously one of the most important, on account of the irritant properties and digestive power of the secretion. On the other hand, the existence of an open sore in the wall of the stomach must itself constitute a source of irritation both to the nerve filaments which it involves and to the other parts of the mucous membrane with which it comes into contact; and since any form of local irritation in the stomach is sufficient to induce a reflex secretion of the gastric juice, it follows that as long

as the disease continues it is apt to be accompanied by hypersecretion. This will be especially the case when the nervous mechanism that controls the pyloric valve is involved in the disease, or food is retained in the organ from obstruction of the outlet. It therefore follows that while hyperacidity is an important cause of the chronicity of an ulcer, the disease itself is usually the cause and not the consequence of the hypersecretion.

9. Associated Diseases

The fact that tubercle of the lung exists in about 17 per cent. of all cases has led to the belief that the pulmonary disease may predispose to the development of the gastric complaint. This, however, is disproved by the results of autopsies made upon persons who have succumbed to phthisis, since Dittrich only observed an open ulcer of the stomach in about 1 per cent. of his cases, while at the Brompton Hospital for Consumption we found that a gastric ulcer only occurred nine times in one thousand consecutive autopsies, or in 0.9 per cent. It has also been suggested that an ulcer of the stomach might lead to the development of phthisis by acting as the seat of local infection, and several cases that have been recorded are claimed to support this theory. But a careful examination of three of these (Buhl, Murchison, Pavne) does not establish the supposition that the gastric affection was the starting-point of the tubercular disease, while in the fourth case (Klebs) the ulcer appears to have been tuberculous from the outset.

It is probable that the frequent association of the two diseases depends upon the fact that a chronic ulcer, like any other serious affection of the digestive organs, interferes with the general nutrition, and thus favours the inception of the infective malady.

A disturbance of the circulation of the stomach induced by diseases of the heart or great vessels is often productive of ulceration. Thus acute ulcers are frequently found in the fundus of the stomach after death from failure of the heart, and appear to originate in the small erosions which ensue from severe congestion of the gastric mucous membrane. Embolic ulcers, on the other hand, are very rare, except in cases of aneurism of the aorta or celiac axis. Chronic ulceration of the stomach may be induced either by atheroma of the vessels of the organ or by chronic congestion of the tissues in long-continued dilatation of the heart. In the former case a localised area of the mucosa becomes insufficiently nourished from the partial occlusion of its nutrient vessel, and gradually falls a prey to the corrosive action of the gastric juice. In the latter the obstruction to the portal circulation diminishes the nutrition of the stomach generally, and thus prevents the repair of any accidental erosion.

Both varieties of ulcer are also encountered in cases of chronic *Bright's disease*, and owe their origin partly to the chronic inflammation of the mucosa which invariably accompanies that complaint, and partly to the failure of the circulation during the later stages of the disease.

Some writers regard syphilis as an important factor in the production of chronic gastric ulcer. Thus Lang states that about 20 per cent. of all cases occur in syphilitic subjects, while others estimate its frequency at 10 to 15 per cent. These assertions have been copied into certain medical works of a popular character, and to our own knowledge have given rise to much domestic trouble, so that it is necessary to scrutinise very carefully the evidence upon which they are founded. Theoretically, the disease may ensue from syphilis in three different ways: (1) by the formation of a gumma in the wall of the organ, which subsequently softens and destroys the mucous membrane; (2) by the production of obliterative inflammation of the gastric vessels (endarteritis), which has the effect of diminishing the blood supply of the stomach, and rendering its tissues vulnerable to the action of the gastric juice; (3) by causing a deterioration of the blood (cachexia), which prevents the repair of any accidental abrasion.

With regard to the first mode of causation, it is to be noticed that gummata of the stomach are extremely rare, even in cases of congenital syphilis (Galliard, Neumann), while the ulcers to which they give rise differ markedly in their general aspect from the simple variety of the disease. Thus, instead of the familiar punched-out ulcer with its clean edges and smooth base, the gummatous variety appears in the form of a deep excavation in the sub-mucous tissue, the edges of which are irregular or overhanging, while its floor is covered with a grey slough or with small cheesy nodules. Again, the mucous membrane in the vicinity of a simple ulcer is either quite

healthy or at most presents signs of reactive inflammation; but in the neighbourhood of the syphilitic disease the tissues are invariably indurated, and not infrequently exhibit numerous small gummata embedded in their substance. Finally, if there is any doubt upon the subject, the microscope will immediately distinguish between a simple ulcer and an ulcerating gumma.

Syphilitic affections of the gastric vessels are rarely encountered, even in cases where the liver and other important organs are extensively diseased. In eighteen cases of tertiary syphilis in which we submitted the stomach to a careful microscopic examination, we were unable to detect any disease of the vascular walls, nor yet in various cases of chronic ulcer occurring in syphilitic subjects have we been able to discover any evidence of endarteritis other than that which usually accompanies the simple form of the complaint. That syphilis may affect the arterioles of the stomach, and give rise to ulceration of its mucous membrane, has been proved beyond doubt (Luxenburg); but the recognition of this truth must not blind us to the fact that the production of a gastric ulcer in this manner is comparatively rare. The clinical observation that certain cases of chronic ulcer of the stomach resist the ordinary methods of treatment, but rapidly improve under the use of mercury or iodide of potassium, is usually supposed to indicate the syphilitic origin of the disease. In some instances this conclusion is doubtless correct, but we believe that the majority are susceptible of another explanation. It is noteworthy that in all the cases of this description the patient is either anæmic or markedly cachectic, and that the gastric symptoms subside pari passu with an improvement in the condition of the blood. It is possible, therefore, that a simple ulcer may form in the stomach quite independently of syphilis, but that its natural tendency to repair may be prevented by the cachectic condition induced by the constitutional complaint.

With regard to the question as to the frequency with which a history of syphilis is met with in cases of gastric ulcer, it is necessary in the first place to distinguish clearly between the two varieties of the disease. The mere fact that acute ulcer of the stomach is almost exclusively confined to young girls seems to preclude the possibility of the specific complaint being an important factor in its ctiology; and this view is

confirmed when the history of such cases is made the subject of special study, for we have never observed a single instance of syphilis among the numerous cases we have investigated.

On the other hand, nearly 10 per cent. of our cases of chronic ulcer had suffered from syphilis at one time or another. This fact, however, loses much of its importance when it is remembered that the subjects of this inquiry belonged to a class which is not only particularly apt to contract the contagious complaint, but is also exposed to those combined influences which render the poor more liable to ulcer of the stomach than the rich. Our experience of diseases of the stomach in private practice has not led us to believe that any undue percentage of persons with a gastric ulcer have suffered from syphilis, nor yet that the subjects of syphilis are exceptionally prone to suffer from it. We therefore think it most probable that in at least one-half of the cases where the two diseases co-exist in the same patient there is no direct relationship between them.

There appears to be an intimate connection between anæmia and gastric ulcer. In 72 per cent. of our cases of the acute complaint in young women there was a definite history of pallor and breathlessness before the first symptoms of the disease manifested themselves; while in a large proportion of the others it was highly probable that the patient had suffered from chlorosis shortly before the occurrence of the hæmatemesis or perforation. We also find that out of 100 cases of severe anæmia in young women who applied for treatment at the London Temperance Hospital, four were definitely affected with ulceration of the stomach, and in seven others the symptoms were highly suggestive of the disease; while among a hundred other girls suffering from various complaints the gastric affection was only diagnosed in two instances.

Several theories have been advanced to explain the relationship between anæmia and ulcer. It has been suggested that the abnormal state of the blood gives rise to thrombosis of a vessel in the stomach which leads to the digestion of a circumscribed area of the mucous membrane. Other writers favour the supposition that the anæmia produces capillary hæmorrhages in the tissues of the viscus, which are subsequently converted into deep erosions; while others again regard the

disease as a result of that altered relation between the arteries and the tissues they supply which, according to Virchow, constitutes one of the principal features of chlorosis. These theories will be discussed in the following chapter, which deals with the pathogenesis of the disease; it is only necessary to remark here that there is reason to believe that anæmia does not so often initiate the complaint as assist in its elaboration by interfering with repair.

When anæmia accompanies chronic uleer of the stomach it is usually the result either of hæmorrhage or general malnutrition.

Disorders of menstruation so frequently accompany ulcer of the stomach in women that some writers have sought to recognise a special variety of the disease under the term 'menstrual ulcer' (Crisp). It appears fairly well established that it is only the acute form of the complaint in young females which exhibits any marked relation to the menstrual functions, since the chronic uleer of later life is not usually associated with any disorder of the catamenia, nor is it particularly common at the climacterie. In our own cases amenorrhea was stated to have preceded the first symptoms of acute gastric ulcer in about 19 per cent.; and to have followed the complaint in 11 per cent.; while in 16 per cent. menstruation had either been irregular or deficient. It is interesting to observe that in nearly all these eases the patient was either anæmic or had previously suffered from chlorosis. In the remaining 54 per cent. the catamenia appeared to be perfectly normal. It is also to be observed that neither women who begin to menstruate at a comparatively late age, nor those who suffer from a sudden suppression of the menses, are unduly liable to gastrie ulcer unless they are also anæmic, while the disease is by no means common during the period of pregnancy. It must therefore be conceded that there is not sufficient evidence to regard amenorrhoea as a primary factor in the production of gastric ulcer.

On the other hand, it is quite certain that even in healthy women menstruation often exercises a remarkable influence upon the digestive functions, the appearance of the catamenia being preceded for a day or two by flatulence, acidity, constipation, loss of appetite, or by some other symptom of disordered digestion. This evanescent disturbance is greatly exaggerated

whenever there exists some functional or organic disorder of the stomach. Thus, a patient who habitually suffers from dyspepsia almost invariably experiences a sudden exacerbation of the complaint just before each period, while in cases of ulcer the pain and vomiting become much more severe, and may lead to complete intolerance of food. It is also interesting to observe that hæmatemesis is particularly apt to occur at these times.

It is obvious, therefore, that the catamenial periods are accompanied by a local disturbance of the digestive organs which not only causes a derangement of their functions, but increases the tendency to hæmorrhage from any ulcerated surface. These facts can only be explained by the supposition that at such times the abdominal viscera become temporarily engorged with blood, the tension of which is subsequently relieved by the discharge. This view is supported by the well-known observation that both the kidneys and the spleen increase in size at the menstrual periods. But severe congestion of the stomach is always liable to be accompanied by small hæmorrhages into the mucous coat of the organ, and occasionally by similar infiltrations of the submucous tissue, which either disappear in a few hours or give rise to multiple erosions. When, however, there exists some constitutional condition, such as anemia, which hinders the repair of the tissues, these insignificant lesions, instead of healing in the ordinary way, may remain for some time or even be converted into progressive perforating ulcers. We believe that the so-called 'acute dyspepsia' or 'gastric catarrh' which occurs at the menstrual periods is really due to congestion of the stomach with erosions of its mucous surface; and that many of the acute ulcers which give rise to hæmatemesis and perforation in young and anæmic women owe their origin to some unusually deep and extensive hæmorrhage into the tissues of the stomach.

Jaksch believed that ulcer of the stomach was exceptionally common after parturition; but more extended experience has not confirmed this statement. It is probable that many of his cases were examples of the acute disease, which is apt to ensue from any septic condition, whether associated with childbirth or otherwise; while in the others the gastric affection had remained latent during the period of pregnancy, and had afterwards undergone a sudden and fatal exacerbation.

Rokitanski attributed some of his cases of gastric ulcer to the influence of malaria, and other writers regard diabetes, lead poisoning, scurvy, general paralysis of the insane, and trichinosis as important factors in the etiology of the complaint, but the evidence which they offer in support of their statements is open to doubt.

CHAPTER IV

PATHOGENESIS

THERE are few subjects in pathology that have given rise to more discussion than the mode of production of a gastric ulcer. Some of the earlier writers, like Abercrombie and Cruveilhier, seem to have been convinced that, as the disease only occurred in the upper part of the digestive tract, it was therefore the result of some special cause, and being accustomed to attribute most of the morbid conditions of the alimentary organs to the effects of inflammation, they naturally regarded catarrh of the stomach as the all-important factor in the formation of an ulcer. Subsequently, Rokitanski showed that a localised necrosis of the mucous membrane invariably precedes the development of an erosion; but, being also imbued with similar ideas concerning the specific origin of the disease, he was led to express the opinion that hæmorrhage into the coats of the stomach was the usual cause of the necrosis. The views of these eminent pathologists have exercised a great influence upon their successors, for we find Leube affirming as recently as 1877 'that gastric ulcer is a specific variety of ulcer which has only one analogue, viz. the corroding ulcer of the neck of the uterus.' On the other hand, Brinton stated his conviction that an ulcer of the stomach was exactly comparable to an ulcer of the leg, both as regards its various modes of production and the chronic course it was apt to pursue.

It is evident, therefore, that the first question that requires to be settled is whether an ulcer of the stomach should be regarded as the outcome of some specific influence, or whether its peculiar features, upon which so much stress has been laid, are merely the result of some secondary and local cause. A little consideration will show that the latter supposition is undoubtedly the correct one.

It is well known that when death ensues from dilatation

of the heart or from such diseases as purpura, scurvy, or septicæmia, it is not unusual to find the whole of the inner surface of the alimentary tract studded with small ecchymoses. In the jejunum and ileum these appear as circular or oval spots of a bright red colour, over which the endothelium remains intact; but in the stomach and the first part of the duodenum they are often replaced by small circular ulcers, whose edges present the dusky stain that is characteristic of their hæmorrhagic origin.

Again, any interference with the blood supply of the stomach or of the upper portion of the duodenum, whether it is caused by the pressure of a tumour upon an artery, or by occlusion of the vessel by embolism or thrombosis, is apt to be followed by a circular perforating ulcer, the dimensions of which vary according to the area of the mucous membrane whose nutrition has been suppressed; but a similar affection of a small branch of one of the mesenteric arteries merely gives rise to that form of infarction which is common to the other abdominal viscera. It is obvious, therefore, that the same cause, operating in different parts of the alimentary canal, is productive of widely different results.

Now we know that the stomach differs from the bowel in being habitually filled with coarse lumps of food, and in the secretion of an acid fluid which rapidly dissolves inert albuminous matter; so it is reasonable to suppose that one or other of these peculiarities may constitute the determinate factor in the production of an ulcer.

With regard to the question of mechanical irritation by the food, it is to be observed that acute ulceration of the stomach frequently occurs during the course of some febrile complaint when the patient is restricted to a liquid diet, and is also met with in infants who have been fed exclusively upon milk, while in the case of the duodenum, whose contents are uniformly semi-solid in consistence, it is only the upper part of the gut which is commonly affected by the disease. We are consequently driven to infer that it is the secretion of the stomach rather than the nature of its contents which converts a necrotic area of the mucous membrane into a definite ulcer.

This inference is amply confirmed both by the teaching of pathology and by the results of experimental research. It is well known that if death occurs when the stomach is empty the organ may retain a perfectly normal appearance for many hours; but that if it ensues soon after a meal, the tissues are quickly digested, and perforation of the viscus may take place from solution of its coats. Again, an acid reaction of the contents of the stomach is absolutely essential to the artificial production of a perforating ulcer in the lower animals. Thus, if interstitial hæmorrhages are induced in the mucous coat by section of the cervical spinal cord, or if the tissue is injured by mechanical or chemical means, the affected parts are readily converted into definite ulcers by filling the viscus with a dilute solution of hydrochloric acid, whilst if the contents of the organ are rendered neutral or slightly alkaline, this change does not take place.

Lastly, it is important to notice that ulceration of the duodenum is chiefly met with in the immediate vicinity of the pylorus, where the mucous surface is exposed to the influence of the acid chyme as it issues from the stomach, and that it is extremely rare in the lower segment of the gut, where the presence of the alkaline secretions of the liver and pancreas are able to neutralise the injurious acidity of the gastric juice. In like manner the œsophagus is only liable to ulceration at its lower end, where its tissues can come into frequent contact with the regurgitated contents of the stomach.

These facts point to the conclusion that there are two distinct stages in the process of ulceration, the first being a local necrosis of the mucous membrane, and the second the removal of the dead tissue; and, since we have found that the latter is ordinarily effected by the solvent action of the gastric juice, the only point to be determined is the usual cause of the necrosis.

Unfortunately this comparatively simple issue has been greatly confused by the exclusive construction that has been placed upon the results of experimental research, for as soon as each new method has been discovered by which an ulcer of the stomach can be produced, it has been immediately proclaimed as the only true mode of origin of the idiopathic disease. Some of the controversies that have been waged upon this subject remind one forcibly of the fable concerning the origin of water, where it was argued that, because the fluid was formed by the condensation of steam, it could not possibly arise from the melting of ice, nor yet by the heating

of snow. Nevertheless all the facts which have been brought to light by experimental research merely prove that the vitality of a portion of the stomach, like that of the skin, can be destroyed in many different ways, some of which are the outcome of disturbed function and others of purely accidental and extrinsic influences. It is therefore necessary to consider the various means by which necrosis of the gastric mucous membrane can be induced, and to decide as far as possible which are most commonly concerned in the production of the lesion in the human subject.

THE CONDITIONS WHICH LEAD TO LOCALISED NECROSIS OF THE GASTRIC MUCOUS MEMBRANE

1. Occlusion of a Nutrient Vessel

A. EMBOLISM

Since Virchow first pointed out the similarity in shape between an ulcer of the stomach and an infarct in the lung or kidney, many experiments have been devised to prove that the disease owes its origin to embolism. Thus, Panum injected an emulsion of wax into the crural artery of a dog against the blood stream, and found that ulceration ensued wherever a particle lodged in a gastric vessel; while Cohnheim, by employing finely powdered chromate of lead, was able to show that the occlusion of even a minute arteriole in the mucous membrane was followed by extensive ulceration. Although these experimental facts are beyond dispute, a brief consideration of the question will suffice to show that embolism of the stomach only occurs under exceptional circumstances, and cannot therefore be regarded as the usual cause of ulcer.

Emboli may be of three kinds: (1) vegetations or clots detached from the left side of the heart or from the inner surface of a large artery; (2) minute clumps of micro-organisms; (3) disintegrated blood corpuscles or masses of pigment.

With regard to the first-named variety, it is to be observed that the gastric arteries usually escape obstruction, even in cases where a large number of emboli are shed from the valves of the heart. Thus, out of 112 fatal cases of ulcerative endocarditis examined at the London Hospital, seventy-one, or

about 62 per cent., presented evidences of embolism in various viscera; but in spite of this the stomach was never once found affected.

Again, it is noteworthy that when artificial emboli are thrown into the circulation, comparatively few ever reach the stomach or duodenum. This fact we have determined by a large number of experiments performed upon dead rabbits, guinea-pigs, and dogs. Immediately after death the thorax was opened, and a cannula tied into the aorta, several of the larger veins being severed at the same time. The vessels were next irrigated with a stream of warm water, which served to expel most of their blood. The cannula was then attached to a pump, and a saline fluid, closely resembling normal blood in density, and containing one hundred emboli, was slowly injected into the aorta. In other cases the fluid was introduced through the medium of a pressurepump, the flow being continuous instead of intermittent, and at a pressure approximate to that which normally obtained in the carotid artery of the animal experimented upon. At the completion of the operation the abdomen was opened, and a careful search made for the emboli. As the result of these experiments we found that only about 3 to 5 per cent. of the emboli introduced into the aorta ever found their way into the gastric vessels, and that of this number more than two-thirds occupied the cardiac or middle zones of the organ; in no instance was the pyloric region or duodenum alone affected.

Similar experiments were subsequently made with tobacco seeds upon the human subject after death, but though as many as five hundred seeds were sometimes thrown into the thoracic aorta, less than 2 per cent. ever found their way into the gastric vessels, and these almost invariably into the arteries of the cardiac end.

When embolism of the stomach does take place, it usually arises from disease of one of the large vessels in the neighbourhood of the organ. In two cases of aneurism of the cœliac axis which have come under our notice, the stomach was found to be profusely studded with ulcers in various stages of development, which doubtless owed their origin to the detachment of minute pieces of clot from the wall of the sac.

It has already been noticed that multiple acute ulcers of the stomach are not infrequent in pyemia and other infective diseases, and since it is known that the blood contains large numbers of micro-organisms in these complaints, it might readily be supposed that the gastric affection is occasionally the result of bacterial embolism. This inference is confirmed by the fact that it is usual to find, along with the ulcers, numerous minute infarcts in the stomach, with evidence of a similar condition in the lungs, skin, and intestines. Microscopic examination also shows that micro-organisms abound in the blood-vessels and tissues in the vicinity of the disease. A



Fig. 30.—Acute ulceration of the stomach in pyemia. Photograph; natural size. (London Hospital Museum.)

beautiful example of this mode of formation of a gastric ulcer is shown in fig. 30, and we have recently seen a case of anthrax in which the stomach and first part of the duodenum were profusely studded with perforating ulcers of similar origin. The statement that the products of blood-disintegration can give rise to obstruction of the gastric vessels is based upon the experiments of Klebs and Welti, who found that broken corpuscles and granules of pigment were sufficient to produce embolic ulceration of the stomach. These facts have been

adopted by London as an explanation of the occasional occurrence of gastric ulcer in cases of malaria, and they may also possibly account for some of the cases which develop during the course of severe anæmia.

It is obvious, therefore, that, except in certain infective diseases, embolic ulceration of the stomach is extremely rare.

B. ENDARTERITIS AND THROMBOSIS

An acute obstruction of a gastric artery by a thrombus is rarely observed except in malignant disease of the stomach. A good example of this has been recorded by Rochemont, where a perforating ulcer developed in the neighbourhood of a cancerous mass, owing to thrombosis spreading from its base. With regard to the significance of hyaline thrombi in the capillaries in gastric ulcer very little can be said. That they are occasionally present is vouched for by so great a pathologist as von Recklinghausen; but the fact that other observers have failed to demonstrate their presence seems to indicate that they are not essential to the formation of an ulcer, and are probably an accidental product of the disintegration of the tissues.

Although obstruction of a gastric artery can only be regarded as an exceptional cause of acute ulceration, it is extremely probable that the gradual obliteration of a nutrient vessel is one of the principal causes of the chronic form of the complaint.

It has already been shown that the inflammatory changes which are so frequently observed in cases of chronic ulcer may either be the result of the lesion in question, or possess an antecedent and independent origin. Of the latter class atheroma of the medium-sized arteries is very common about middle age, when ulceration is most apt to occur, while Luxenburg and others have demonstrated that syphilitic endarteritis sometimes gives rise to chronic ulcer in young adults. It is also certain that the superficial chronic ulcers met with in the pyloric region in cases of phthisis are usually due to lardaceous disease of the vessels (Merkel, Edinger). In all these cases the gradual diminution in the supply of arterial blood produces malnutrition of the mucous membrane, and thus deprives it of its natural power of resistance

to the corrosive action of the gastric juice. In this manner diseases of the gastric vessels both predispose to the formation of an ulcer and also render the disease, when once it has commenced, incapable of rapid healing.

C. VENOUS OBSTRUCTION

Ligature of the portal vein was found by Müller to give rise to ulceration of the stomach, and this discovery is supposed to prove that a gastric ulcer may be caused by an obstruction to the venous circulation through the organ. There are, however, several facts which are directly opposed to this 'congestion theory' of the disease.

The gastric veins are entirely destitute of valves; and as they form an intimate and complex anastomosis over the whole mucous membrane of the organ, the venous system can be readily injected or emptied through the medium of any one vein. As a result of this, acute local congestion is almost an impossibility, since any sudden impediment to the flow of blood through one vein would lead to its immediate determination through a neighbouring channel. Indeed, if it were not for this free anastomosis, every contraction of the pylorus, by compressing the veins which pass obliquely through its muscular structure, would lead to serious mischief in the adjacent mucous membrane. With this fact in mind, it would appear probable that the obstruction of a small venous tributary is incapable of producing any serious effect upon the gastric tissues. In order to test the accuracy of this view, the following experiments, among others, were undertaken:-

Experiment 1.—A healthy rabbit was anæsthetised, and the abdominal cavity opened in the median line. The stomach was then drawn downwards so as to expose the structures in the lesser omentum, and ligatures were applied to two small gastric veins close to the stomach, and before their junction with the portal vein. The wound was closed with all antiseptic precautions. The animal was killed at the end of twenty-four hours, and although the ligatures were found to have completely occluded their respective veins, no sign of hæmorrhage or undue congestion could be discerned in the neighbouring regions of the mucous membrane.

Experiments 2 and 3.—Two other rabbits were treated in

a similar manner, one being killed in six hours, and the other at the end of two days. In both instances the stomach was found to be perfectly normal, and even with the microscope no disease of the mucous membrane could be detected.

Experiment 4.—A similar experiment was performed upon a cat, three small veins, one near the lesser and two at the greater curvature, being securely ligatured. The animal was killed at the end of ten days, but nothing abnormal could be detected in the stomach.

The next point for consideration is the effect of sudden impediment to the whole or greater portion of the venous outflow from the stomach.

Experiment 5.—Two healthy rabbits were selected, and in both the portal vein was ligatured just outside the portal fissure. The first animal was killed six hours after the operation. The inner surface of the stomach was found to present numerous hæmorrhages in the mucous and submucous tissues, which varied in size from a pin's head to a small bean, and in some instances were the seat of ulceration. These hæmorrhages were almost entirely confined to the cardiac and middle thirds of the organ, only two minute extravasations being found in the pyloric region. The second rabbit succumbed about eighteen hours after ligature of the portal vein. In this case the fundus of the organ exhibited numerous small circular ulcers, the edges of which formed a tumid ring, while their floors showed unmistakable signs of the hæmorrhagic origin of the disease. Here again the pyloric region was but slightly affected.

Experiment 6.—In another rabbit the main trunk of the portal vein was left untouched, but the various gastric veins entering it or its large tributaries were carefully secured. The animal was killed at the end of forty-eight hours, and the stomach was found to present numerous hæmorrhagic ulcerations in the fundus and in the region of the great curvature. The veins near the cardiac orifice were enlarged and very turgid. The mucous membrane in the pyloric region was also slightly affected.

Experiment 7.—Abdominal section was performed on a healthy rabbit, and 10 minims of tincture of perchloride of iron carefully injected into the portal vein. The animal was found dead on the following day (twenty-two hours). At the autopsy

the fundus of the stomach exhibited numerous hæmorrhages, many of which had undergone superficial digestion. Only a few extravasations were found on the posterior wall of the pyloric region.

From these and other similar experiments two conclusions may be drawn. In the first place, sudden obstruction of a small vein in the stomach is unattended by any deleterious consequences, the local congestion of the mucous membrane which ensues being instantly relieved by drainage into the surrounding vessels. Secondly, acute obstruction of the portal vein is followed, as Müller showed, by hæmorrhages into the mucous membrane, which frequently proceed to ulceration. But these morbid appearances are multiple, and almost exclusively confined to the cardiac and central regions of the stomach, and therefore differ in a very noticeable manner from the ordinary solitary perforating ulcer.

D. VASCULAR SPASM

The view advocated by Klebs, that ulceration of the stomach depends upon local spasm of the blood-vessels, is supposed to have received a certain amount of corroboration from some experiments performed by Talma, who showed that when the left vagus nerve is faradised, tonic spasm of the pylorus results. If this electrical excitation be maintained for some time (two to six hours), and the animal be killed a few hours later, ulcers of various sizes and shapes may be found occupying the pyloric region of the stomach. The explanation of this phenomenon is, however, a simple one. The arterial trunks in the pyloric region have to pass obliquely through a thick layer of muscular tissue in order to reach the mucous membrane; hence a tetanic contraction of the gastric wall must compress the blood-vessels, and, by rendering the mucous membrane anæmic, permit its erosion by the gastric juice. As the result of his experiments, Talma concludes that pyloric spasm plays an important part in the production of acute perforating ulcer in young adults, and in support of his theory he quotes three instances where intense abdominal spasm was succeeded by hæmatemesis. But this premonitory symptom has hitherto escaped the observation of English physicians, for the abundant literature upon the subject is

singularly silent upon this essential point; and among our 383 clinical cases of gastric ulcer we can find no notice of any initial symptom akin to that described by Talma. Indeed, it is probable that the majority of clinicians would be disposed to regard abdominal spasm in such a case as dependent upon an overloaded state of the colon, a condition not rarely encountered in this disease. Tetanic contraction of the pylorus, as a common cause of acute gastric ulcer, must therefore be set aside, until it can be explained why the left vagus nerve should once in a lifetime, and for the space of three or four hours, be liable to tetanic excitation without the exhibition of the ordinary symptoms of severe muscular spasm.

E. DIRECT PRESSURE

Necrosis of the gastric mucous membrane may be brought about by direct pressure upon the wall of the viscus. In the records of the London Hospital two cases of aneurism of the abdominal aorta are recorded, in which the wall of the sac became adherent to the posterior aspect of the fundus, and gave rise to a large ulcer in this region of the organ. In one instance a thick cord of omentum, one end of which was attached to the liver, and the other to the wall of the pelvis. was found to have constricted the pyloric half of the stomach, and to have produced a deep linear ulcer on its inner surface corresponding to the line of pressure. In another, chronic peritonitis of tuberculous origin had occasioned such enormous thickening of the coats of the stomach that the organ was reduced to about one-quarter of its normal size, while almost the whole of its mucous surface was in a state of simple ulceration. Cases have also been recorded where a gallbladder filled with calculi, or a tumour of the liver, pancreas, or kidney, was the cause of the 'pressure necrosis'; and Netter has related one in which a calcified cyst of the liver produced a large ulcer of the stomach by its pressure upon a branch of the coronary artery. Lastly, it may be mentioned that fibromyomata in the submucous tissue of the organ occasionally induce necrosis of the superimposed layer of mucous membrane in a similar manner.

2. Hæmorrhage into the Walls of the Stomach

Hæmorrhagic infiltrations of the inner coat of the stomach occur under many conditions. In the human subject they are found after death from dilatation of the heart or from acute obstruction of the portal circulation, in disorders of the blood. such as purpura, scurvy, and severe anæmia, and in the malignant specific fevers. Experimentally they have been produced in the lower animals by injuries inflicted upon the central nervous system, such as section of the thalami and cerebral peduncles (Schiff), destruction of the anterior corpora quadrigemina (Ebstein, Brown-Séquard), and division of the cervical spinal cord. In all these cases erosions are apt to ensue from the action of the gastric juice upon the dead tissue, and Koch and Ewald were able to produce deep perforating ulcers by filling the stomach with a weak solution (5 per cent.) of hydrochloric acid. Rindfleisch and Axel Key adduced these facts to explain the mode of origin of the idiopathic disease, and supposed that a spasmodic contraction of the pylorus took place during attacks of vomiting or gastralgia which so compressed the veins traversing the muscular coat as to induce localised extravasations of blood. This elaborate hypothesis merely shows that its authors possessed imaginative faculties of a high order, as there is no evidence to show either that the pyloric end of the stomach is subject to attacks of tetanus, or that such contraction can give rise to hæmorrhagic infarctions.

3. Inflammation

Severe inflammation may give rise to necrosis of the gastric mucous membrane, either by a process of sloughing or through the medium of interstitial hæmorrhage. The former condition is rarely encountered, and is only noticed three times in the pathological records of the London Hospital. In two of these cases the patient succumbed to disease of the kidneys, and several sloughing ulcers were observed in the cardiac end of the stomach, and in the intestine near the ileo-cæcal valve, while in the third instance, where death had occurred from gangrene of the face, several necrotic patches with adherent sloughs were found in the region of the great curvature. A somewhat similar case to the last has been recorded by Klebs.

PATHOGENESIS 109

The hæmorrhagic infiltrations of the mucous membrane which frequently accompany acute inflammation of the stomach are occasionally converted into penetrating ulcers of considerable size. They differ, however, from the ordinary form of gastric ulcer in that they are usually multiple, and affect the fundus and the greater curvature of the organ rather than the pyloric region and the upper border. It is also to be observed that in the simple perforating ulcer both the edges of the sore and the surrounding tissues are free from the microscopical signs of inflammation, while in the chronic variety of the disease the inflammatory thickening of the ulcer is merely a secondary manifestation of the complaint.

Although these facts render it improbable that inflammation is the ordinary cause of ulceration of the stomach or duodenum, there is reason to believe that that form of the complaint which ensues from superficial burns and other septicæmic conditions may owe its origin to severe catarrh of the mucous surface.

Since Curling first drew attention to the occasional association of duodenal ulceration with superficial burns, various theories have been formulated to explain the connection between the two morbid conditions. At the present time there seems to be an inclination to ascribe the intestinal disease to pyæmic embolism. There are several facts, however, which militate strongly against this supposition. It has already been shown that when artificial emboli are introduced into the general circulation, the stomach and duodenum are rarely affected; and even when their vessels do become occluded, embolism of the duodenum never occurs without a similar affection of the stomach. But when ulceration ensues from burns of the skin, the lesion is much more common in the duodenum than in the stomach, so that if the embolic theory of the disease is accepted, it is necessary to assume that there exists some special mechanism whereby the microorganisms in the circulation are directed into the vessels of the first part of the intestine in preference to those of the stomach. This assumption is negatived, however, by the fact that in ordinary cases of pyæmia secondary ulceration of the digestive canal is more common in the stomach than in the duodenum, the ratio, according to the London Hospital records, being about five to one.

Again, if embolism were the usual cause of the disease, it

might be expected that in some cases, at any rate, the occluded vessel would be found on microscopical examination of the tissues. As far as we know, however, the supposititious block has never yet been satisfactorily demonstrated.

On the other hand, the one constant and prominent feature of the alimentary canal after death from burns is acute inflammation of its mucous membrane. This inflammatory

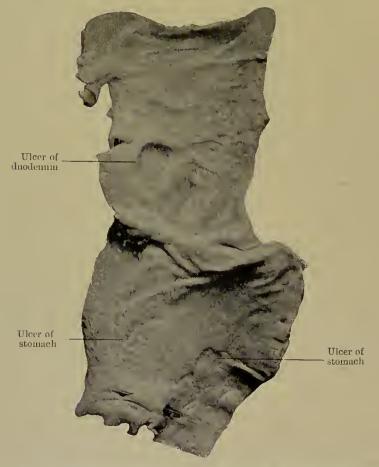


Fig. 31.—Photograph of a portion of the stomach and duodenum after death from burns, showing acute ulceration in both organs. Natural size. (London Hospital Museum.)

condition varies greatly in intensity in different cases; in some being so slight as to require the microscope for its detection, while in others it is so intense that it involves the whole thickness of the bowel, and may even give rise to peritonitis. In almost all cases, however, it may be observed that the maximum intensity of the inflammation is situated at the upper part of the small intestine, and is apt to be associated

with mucous and submucous hæmorrhages and enlargement of the solitary glands. Moreover, in three cases of superficial ulceration which have come under our notice microscopical examination showed that the loss of tissue had been preceded by a hæmorrhagic infiltration of the mucous membrane associated with acute inflammation of its substance. It is highly probable, therefore, that ulceration of the duodenum and stomach from burns is merely a secondary and more or less accidental result of the gastro-duodenal catarrh which so frequently ensues from the injury to the skin.

Hunter has recently shown that duodenitis accompanied by ulceration can be produced in dogs by the subcutaneous injection of toluvlidiamine, and has suggested that the analogous lesion met with in burns is caused by the elimination by the bile of poisonous substances absorbed from the injured surface, which are thus brought into direct contact with the mucous membrane of the bowel. It is a well-established fact that the liver is constantly excreting organic poisons from the circulation, but it is hardly permissible to assume that in the exercise of this normal function it habitually jeopardises the structural integrity of an important neighbouring organ like the intestine. Moreover, it is to be observed that, although the signs of internal inflammation after death from burns are usually most severe in the duodenum and ileum, the other portions of the alimentary tract also suffer in a similar manner, while not infrequently ulcers develop in the stomach, which is beyond the influence of the irritant bile. We may also add that in repeating Hunter's experiments we were able in one case to produce two perforating ulcers in the duodenum after a preliminary ligature of the bile duct, so that it is obvious that the inflammatory lesion cannot be entirely due to local irritation by the toxic bile.

The digestive and absorptive functions of the alimentary canal are of such paramount interest and importance that the eliminative powers of the gastro-intestinal mucous membrane are apt to be forgotten. It is clear, however, both from experimental research and clinical knowledge, that the stomach and intestine possess vicarious excretory functions of an important character. Binet has shown that certain substances, such as iodide of potassium, the salts of magnesium and lithium, and many of the alkaloids, are very readily climinated

by the stomach, while strontium, antimony, and other drugs cannot be detected in the contents of the organ after their subcutaneous injection. In like manner the various portions of the intestine are capable of eliminating special substances when the ordinary channels of excretion happen to be obstructed. It is also a curious fact that the salts of iron, which are not usually absorbed from the stomach, are rapidly eliminated by the gastric mucous membrane.

It is not, however, so generally known that the exercise of this vicarious function is apt to induce inflammation of the tissues. This important fact was brought to our notice some years ago, when we were engaged in investigating the origin of the gastro-enteritis of kidney disease; for we then found that the subcutaneous injection of urea was not only followed by the appearance of the salt in the contents of the stomach and intestines, but that its frequent employment gave rise to acute inflammation of the mucous membrane of these organs. The same results attend the injection of cantharidin and other drugs (Aufrecht).

It would therefore seem that whenever the alimentary canal is called upon to aid in ridding the system of a toxic substance, whether this has been produced by the action of micro-organisms in the blood (specific fevers, pyemia, &c.), or has merely been absorbed from the surface, it is extremely apt to become inflamed as the result of its abnormal activity, the severity of the inflammation varying in different parts of the tract according to the nature of the poison. We have endeavoured to corroborate this theory in cases of burns, by collecting the sloughs from the surface and immersing them in absolute alcohol for some weeks, after which the soluble proteid material was extracted with distilled water, and reprecipitated by alcohol. In this manner a fine white powder was ultimately obtained, which gave most of the reactions of an albumose, and when injected beneath the skin of a cat or rabbit gave rise to an appreciable degree of duodenitis. Unfortunately the supply of material was too small to permit of more conclusive experiments, but we are strongly of opinion that further investigations in this direction may throw considerable light upon the origin of secondary gastro-intestinal inflammations.

4. Mechanical Injuries

Cuts or abrasions of the mucous coat of the healthy stomach which do not inflict any serious amount of damage upon the surrounding parts, such as are produced by swallowing sharp instruments or by careless manipulation of the stomach tube, invariably heal rapidly and completely (Daettwyler, Colmheim); when, however, the injury is of such a kind as to cause hæmorrhage into, or inflammation of, the subjacent structures, repair is tardy and is often preceded by ulceration. The best example of traumatic ulceration of the stomach is to be found in cases where continued pain and hæmatemesis follow severe blows upon the epigastrium, for Ritter and Vanni have shown by experiment that such injuries may give rise to a hæmorrhagic infiltration of the coats of the viscus with subsequent exfoliation of the dead tissue.

5. Chemical and Thermal Injuries

The direct application of corrosive fluids to the surface of the stomach produces either immediate sloughing or intense inflammation of the affected parts, according to the degree of irritation excited. In either case ulceration is apt to occur and to persist for a considerable time, more especially in the neighbourhood of the pyloric and cardiac orifices. Decker has also shown that the introduction of hot food into the stomach of a dog may give rise to interstitial hæmorrhages which subsequently develop into perforating ulcers. It is extremely doubtful, however, whether the disease ever arises in the human subject from such a cause (p. 85).

6. Neurotic Conditions

The enormous influence which the nervous system exercises upon the gastric functions has led to the belief that ulceration may be due to a disturbance of the nervous mechanism of the stomach. It is conceivable that an ulcer might arise from this cause in three different ways, namely, by an increase of blood-pressure, by a disturbance of trophism, or by an alteration in the secretion of the organ.

The experiments of Schiff, Ebstein, and Ewald have shown

that injuries inflicted upon the brain and spinal cord are apt to produce punctiform hæmorrhages in the mucous coat of the stomach, which subsequently develop into definite ulcers. In such cases, however, the lesion is invariably multiple and more often affects the fundus than the region of the lesser curvature, while the fact that the sufferers from the idiopathic disease seldom exhibit any symptoms of a nervous lesion is sufficient to exclude this mode of origin from the domain of practical discussion.

The theory of trophic disturbance was brought forward by Wilks and Moxon, who sought to compare a simple ulcer of the stomach with that form of ulceration of the cornea which occasionally follows paralysis of the trigeminal nerve. There are reasons for thinking, however, that this lesion is not wholly independent of the co-existing condition of anæsthesia; while in the case of the stomach all our knowledge goes to show that the sensibility of the mucosa is exalted rather than diminished. This theory is entirely speculative, and has never gained acceptance.

Osborne was the first to suggest that an ulcer might arise from the secretion of an abnormally acid juice by a circular group of the gastric glands, and more recently Günsburg has extended this theory by attributing the disease to a hyperacid state of the gastric secretion brought about by abnormal nervous influences. There is no doubt that the majority of the cases of chronic ulcer are attended at some period or other by an excessive acidity of the gastric secretion; but this phenomenon is not constant, and is usually absent in the acute variety of the disease, while not infrequently ulceration takes place when the acidity of the gastric contents is greatly diminished (Luxenburg). It is also noteworthy that ulceration is rarely encountered in cases of functional hyperacidity. Until, therefore, these anomalies can be satisfactorily explained it cannot be accepted that an excess of acid in the gastric secretion is invariably the primary factor in the production of the malady.

7. Bacterial Necrosis

The important part played by bacteria in the causation of disease has naturally led to the belief that ulceration of the stomach may possibly owe its origin to micro-organic activity.

This view seems to have been first promulgated by Boettcher, who discovered micro-organisms in the edges of an ulcer, and has more recently been advocated by Letulle, Nauwerck, and In support of their contention these latter Schmilinsky. writers lay considerable stress upon the occurrence of gastric ulcer in cases of pyamia, anthrax, and other infective disorders. and also after injections into the peritoneal cavity of pure cultures of the Staphylococcus pyogenes and other microbes. These arguments, however, have no bearing upon the present point, since it has already been shown that in pyæmia the ulceration usually ensues either from embolism or from acute inflammation induced by the presence of toxins in the blood. The question with which we are now concerned is whether any bacteria present in the contents of the stomach are able to produce a non-inflammatory necrosis of the mucosa akin to that which occasionally occurs in guinea-pigs and other lower animals. As far as we know, the only circumstance which appears to support this belief is the occasional discovery of micro-organisms in the edges of an ulcer and in partially detached sloughs; but since all necrosing tissues are rapidly invaded by bacteria, the phenomenon does not constitute any evidence of the mycotic origin of the disease.

On the other hand, it is well known that the stomach possesses a high degree of immunity from local infection, owing to the small quantity of lymphoid tissue in its walls, and to the antiseptic properties of its acid secretion. The importance of the former factor is shown by the rarity of tubercular affections of the viscus, and of the latter by the experimental investigations of Sieber, Miguel, Kast, Bunge, Minkowski, and others. It is also probable that the healthy mucous membrane possesses a natural tendency to resist the invasion of micro-organisms, since it has been proved that wounds and abrasions of the tissue, when examined immediately after death, never exhibit any signs of bacterial invasion (Leith). It is therefore obvious that if ordinary ulceration of the stomach is due to bacterial necrosis, it must either arise from some special form of infection, or from the temporary inhibition of the antiseptic properties of the gastric juice. With regard to the former supposition, it should be observed that no specific organism has ever been recognised, even by the most ardent supporters of the mycotic theory. Again, the disproportionate frequency of the disease among the female members of a family renders it highly improbable that infection can take place through the medium of the food; while the practical immunity which is enjoyed by infants and young children, who in all other respects are the greatest sufferers from gastro-intestinal infection, constitutes a negative fact of great importance.

But even if it be allowed that the stomachs of young women are apt to become infected by a species of microorganism which is capable of exercising a deleterious influence upon the inner surface of the organ, is it not extraordinary that the bacteria should habitually concentrate their power upon one, or at most two, minute spots, instead of giving rise to a multiple series of inoculations such as occurs in tubercular affections of the intestinal tract; and also that they should exhibit such a remarkable proclivity for the comparatively inaccessible region of the lesser curvature rather than for the fundus of the organ, where a local irritant always produces its most noticeable effects?

The supposititious diminution of the acidity of the gastric juice is not borne out by the results of actual investigation, since not only are the majority of chronic ulcers accompanied by a condition of hyperacidity, but many authorities affirm that chlorosis and amenorrhoa, which are supposed to possess an etiological relation to the disease, are themselves accompanied by an abnormally acid state of the gastric secretion (Einhorn). Martin has endeavoured to overcome this difficulty by supposing that the bacteria gain access to those gastric glands which do not secrete hydrochloric acid, and points to the pyloric situation of the chronic ulcer as a fact which corroborates this view. Unfortunately, however, the acute form of the disease happens to be especially common in that region of the viscus where the glands are most actively engaged in the formation of the acid, and the most imaginative mind can hardly picture a number of microbes patiently waiting for the intervals of digestion to make their deleterious onslaught upon the tissues. Until these difficulties have been explained, the theory of 'bacterial necrosis' will not find any wide acceptance.

8. Diseases of the Solitary Glands

Although it has several times been suggested that the solitary glands of the stomach might be responsible for some

forms of ulceration, this possible mode of origin has attracted little attention. It was formerly denied that the stomach possessed any circumscribed masses of lymphoid tissue; and it was not until the publication of the researches of Handfield Jones, Samuel Fenwick, Fox, and Shäpfer, that their existence was placed beyond dispute. These observers demonstrated that the stomachs of most mammals present numerous collections of lymphoid tissue around the fundi of the secreting tubules, which vary in size in different parts of the organ, being largest and most numerous in the pyloric region near the lesser curvature. It was also shown that these solitary glands become enlarged in the various inflammatory disorders of the stomach, and occasionally give rise to distinct ulceration of the mucous membrane.

The importance of the subject may serve as an excuse for a more detailed account of the anatomy and pathology of these lymphoid structures.

When the stomach is taken from a recently killed pig, and the mucous membrane carefully peeled off from the subjacent muscular structures, numerous small hemispherical masses, varying in size from a pin's head to a hempseed, may be seen upon its deep surface. If the mucous membrane is now immersed for some hours in a dilute solution (one in ten) of glacial acetic acid, it swells up and becomes gelatinous and transparent, while at the same time the solitary glands appear swollen and opaque, and thus become recognisable in places where previously they had been incapable of detection. These structures may also be demonstrated by the use of hydrochloric instead of acetic acid. Xylol renders the whole tissue transparent, and if the immersion be stopped at the right moment the solitary glands can be readily seen by transmitted light. By any of these means it is easy not only to determine the presence of solitary follicles in the mucous membrane of the stomachs of various animals, but, what is of even greater importance, to estimate their relative number in any particular region of the organ. Next to the pig, the cat and the child appear to be endowed with the most elaborate system of solitary glands, while in the rabbit we have never been able to detect any.

In order to determine, as far as possible, the relative number of the solitary glands in the various regions of the organ,

numerous specimens were obtained from the post-mortem room as soon after death as possible, and treated with dilute acid in the manner described. The surface of the mucous membrane was then mapped out into areas of a square centimetre, and the number of follicles counted in each, a mean of the observations representing their average frequency. As the result of these investigations several interesting facts have come to light, which amply confirm some of the previous statements of Handfield Jones. It would appear that in the human subject the solitary glands of the stomach vary both in size and number at different periods of life. In infancy and childhood these collections of lymphoid tissue are fairly developed and profusely scattered over the whole surface of the organ, but are always particularly large and distinct in the pyloric region near the lesser curvature, this being the spot to which most of the lymphatic vessels direct their course on their way toward the lesser omentum. The actual number of glands varies enormously in different cases, but the following may be taken as a rough representation of the mean of a large number of observations. Before the age of seven months the glands are generally small and ill-defined, and can be best demonstrated in the region of the lesser curvature. Between the ages of one and ten years the solitary follicles are numerous and of some size, numbering in the cardiac zone from three to seven in each square centimetre area, and rather less in the middle third of the organ. In the vicinity of the lesser curvature in the pyloric region, we have often counted as many as eight to fifteen in an area of the same size. From puberty onwards the solitary glands undergo a gradual diminution, and seem to retreat from the cardiac extremity toward the lesser curvature. Thus, in young adults we have often been able to discover only one or two glands in two square centimetres of the fundus of the organ, though in the pyloric region the deficiency was much less marked. After the age of forty it is often impossible to demonstrate their presence in the cardiac two-thirds of the stomach without the use of the microscope, and in the pyloric region they are much reduced in number, and appear small and shrunken. In some of these cases one is often struck by a peculiar pitted appearance of the mucous membrane, and on submitting this to the microscope it becomes evident that the pit-like depression is the result of a retrogressive change in

a solitary gland. Occasionally the lymphoid tissue undergoes a form of hyaline degeneration.

Microscopic examination reveals two forms of lymphatic structures in the stomach. In the simplest variety the tissue consists of a few nucleated cells, situated around the blind extremities of the gastric tubes, and spreading over the muscularis mucosæ in the form of a thin layer. In inflammatory disorders of the mucous membrane these diffuse collections of lymphoid tissue undergo a rapid increase in size, and often completely obscure the deeper portions of the secreting structures.

In the second variety the lymphoid tissue exists as circumscribed masses in the substance of the mucous membrane, and constitutes the solitary glands already referred to. In the human subject this gland or 'follicle' is usually somewhat oval in shape, and rests upon the muscularis mucosæ like an eggshell upon its broken end. At the circumference of the mass the gastric tubules are displaced and assume a slanting direction, but towards the surface they regain their perpendicular position, and completely cover in the superficial aspect of the gland. No limiting membrane can be discovered, and the lymphoid elements of the follicle often encroach slightly on the surrounding tissues. Each solitary gland is provided with a small nutrient artery, and a lymphatic vessel passes from its deeper surface obliquely through the muscularis mucosæ to join the larger trunks situated in the submucosa. It occasionally happens that the base of the gland is situated in the submucous tissue, and extends thence into the mucous membrane through a gap in the muscularis mucosæ, the whole mass resembling an hour-glass in outline. This variety we have only encountered in the pyloric region, near the lesser curvature.

Like their homologues in the intestinal tract, the solitary glands of the stomach are prone to become the seat of disease, but owing to their insignificant size and deep situation in the mucous membrane they possess a far greater degree of immunity than is accorded to the Peyer's patches and solitary glands of the intestine. For this reason the stomach is rarely attacked by tuberculous ulceration, though it must be noticed that when this does occur the disease is usually found where the lymphatic tissue is most abundant, namely, in the pyloric region near the lesser curvature.

The solitary glands of the stomach are frequently found to be enlarged in cases of Addison's disease and lymphadenoma, but in these necrotic changes are seldom observed. On the other hand, in the various inflammatory conditions of the stomach, and especially in infective disorders, such as typhoid fever, acute tuberculosis, and diphtheria, these glands exhibit signs of acute inflammation, and their tissue undergoes rapid necrosis. The earliest signs of this condition consist in a proliferation of the lymphoid elements with a corresponding increase in the size of the follicle, and distension of its vessels. These changes are best observed in a case of gastritis, when it is found that the mass loses its definite outline, and tends to spread in all directions. In fig. 32, the tubules in the neigh-

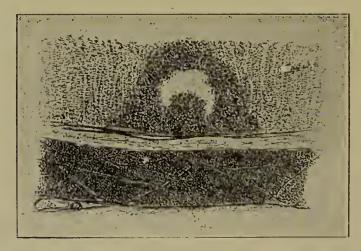


Fig. 32.—Section of a human stomach affected with acute catarrh, showing enlargement of a solitary gland (the centre of the mass has fallen out).

bourhood of the follicle are seen to be pressed upon and partially obscured by the lymphoid cells, which can be traced for some distance from the inflamed follicle. It will also be noticed that the portion of the mucous membrane superficial to it is densely infiltrated with cells, and all signs of the tubules are obliterated. The next stage in the disease is marked by an extension of the process of cell-infiltration, accompanied by changes in the centre of the gland, the cells of which lose their individual outlines and undergo rapid degeneration, with the result that in many instances a small cavity is formed in the centre. This necrosis spreads in a peripheral direc-

tion; the gastric tubes become twisted and obliterated by the increasing pressure, and their fundi are often converted into small cysts. The same degenerative changes occur in

the superficial portion of the mucous membrane, which is thinned out by pressure from below until it finally gives way, and the semi-fluid contents of the follicle are discharged into the cavity of the stomach.

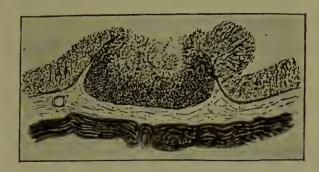


Fig. 33.—Section of a stomach (guinea-pig) affected with follicular ulceration.

About the time that the gland is undergoing softening, it is generally found that the muscularis mucosæ has suffered considerably from round-cell infiltration, and in some instances the follicle appears to have passed through the muscular layer

and to have established its base in the submucosa. In the guinea-pig, owing to the peculiar shape of the follicle, and to the fact that it lies entirely beneath the muscularis mucosa, a portion of the mucous membrane often becomes included in the lymphoid tissue,



Fig. 34.—Section of a stomach (guinea-pig), showing an enlarged solitary gland in the submucous tissue, with inversion of the mucous membrane (× 40).

where it undergoes rapid degeneration (fig. 34).

From these facts it is obvious that inflammation attacking the solitary glands may terminate in actual ulceration of the unicous membrane. In children who have died from acute tuberculosis, the stomach sometimes presents numerous circular ulcers, one or two millimetres in diameter, scattered over the whole surface of the organ. These seldom extend deeper than the submucous tissue, and present slightly tumid edges surrounded by a zone of congestion. The microscope demonstrates that they have originated in the

lymphoid structures, and are not due to the softening of miliary tubercle. This form of ulceration has been described by many writers, and among ten cases of acute tuberculosis we examined at the Evelina Hospital, four presented the appearances narrated.

It might easily be imagined that since acute tuberculosis is an infective fever, other examples of this type of disease



Fig. 35.—Drawing of the pyloric end of the stomach in a case of enteric fever; a, acute perforating ulcers with clean bases; b, an ulcer with adherent slough.

might be accompanied by similar lesions in the stomach. In scarlatina, measles, erysipelas, and small-pox, morbid changes are invariably found; but it is the secretory structures that primarily suffer, and not the solitary glands. On the other hand, in three cases of diphtheria which we recently examined, distinct follicular ulceration was observed in the pyloric region. But the most interesting point in this connection is the occasional occurrence of ulceration of the stomach in cases of enteric fever.

We recently obtained a number of stomachs from guineapigs which had died after inoculation with pure cultures of Eberth's typhoid bacillus. In five instances out of fourteen, well-marked circular ulcers were found occupying the pyloric region of the organ near the lesser curvature, which presented all the features characteristic of ulceration of the solitary glands. By means of the microscope small masses of lymphoid tissue were discovered scattered throughout the viscus, and the

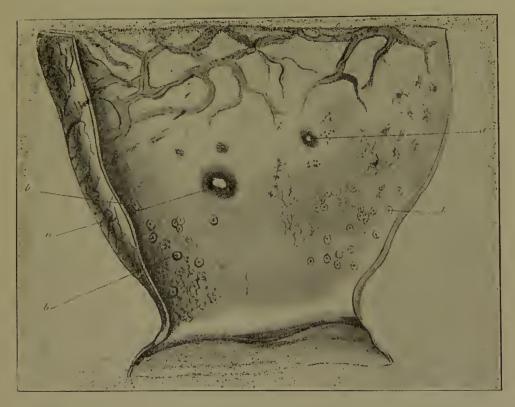


Fig. 36.—Drawing of the pyloric end of the stomach, showing inflammation of the solitary glands, with acute ulceration originating in these structures. (After Carswell.) a, perforating ulcers; b, inflamed solitary glands.

various stages in the process could easily be traced. Acute ulceration of the stomach also occurs in cases of enteric fever affecting the human subject, and several cases of this kind have been placed on record. In fig. 35, which is taken from a specimen in the museum of the London Hospital, three well-defined circular ulcers are situated in the pyloric region of the stomach, one of which presents a semi-detached slough. The largest of them measured 5 mm., and the smallest 3 mm. in diameter. The edges were thin, and in

one case distinctly undermined, the bases being formed by the submucous or muscular layers. It appears probable that the gastric juice in this case was too weak to digest the necrotic tissue, whence the thin undermined edge of the ulcer and the separation of the entire slough. On microscopic examination the lymphoid tissue of the stomach was found to be enormously increased in amount, and the supposition that the ulcers originated in disease of the solitary glands was easily confirmed. It may also be mentioned that one of the ulcers presented small blood clots attached to its sides and base. The microscope showed that the hæmorrhage was purely superficial, and had probably ensued upon the separation (digestion?) of the slough.

That inflammation in these structures may give rise to typical perforating ulcers is also well shown by an illustration in Carswell's 'Atlas of Pathology,' where a drawing of the pyloric end of the stomach exhibits the successive stages in the process (fig. 36).

It is obvious therefore that under certain conditions inflammation of the solitary glands may give rise to ulceration of the stomach, which, by extending its area in all directions, can assume the characteristic form of an acute perforating ulcer.

The Elaboration of the Ulcer

Although it is evident from the facts which have just been cited that an ulcer of the stomach may be produced in many different ways, there yet exists one important distinction between the disease when artificially induced and that which is developed spontaneously in the human subject; for it is found that the former heals rapidly and completely, while the tendency of the latter is to progress. We are thus brought face to face with what is really the chief problem in the pathogenesis of the disease, namely, the determination of the various conditions which hinder the repair of an abrasion of the gastric mucous membrane. These may be either local or general.

LOCAL CONDITIONS WHICH INTERFERE WITH THE REPAIR OF A GASTRIC ULCER

1. The Influence of Situation

Among the manifold differences which exist between the acute and chronic forms of the disease, the anatomical situation of the ulcer is by no means the least remarkable. The acute variety exhibits a special predilection for the cardiac and central portions of the stomach in the vicinity of the lesser curvature, while the chronic form of the complaint usually develops in the neighbourhood of the pylorus. Again, persons who survive the immediate effects of swallowing corrosive fluids, like nitric or hydrochloric acid, not infrequently succumb, after the lapse of several months, to tuberculosis or some other indirect result of the gastric injury; and in such cases it is interesting to observe that although the greater part of the mucous membrane has completely healed, a chronic ulcer is often found to exist near the pyloric orifice. Lastly, it can be shown by experiment that mechanical and other forms of injury inflicted upon the mucous membrane in the region of the pylorus heal much more slowly than those which affect the fundus of the organ. The only conclusion, therefore, which can be drawn from these facts is that certain special conditions exist in the pyloric portion of the stomach which retard the process of tissue-repair at this spot. These several conditions appear to be as follows:--

- (a) Want of rest.—It is well known that one of the chief obstacles to the healing of a wound situated near a joint is the constant movement of the surrounding tissues. In surgical practice this injurious influence can be controlled by the application of a splint to the affected part, but in the case of the stomach it is impossible to prevent the constant contraction and relaxation of the pyloric sphincter, or to paralyse the peristaltic movements of the organ. Want of rest, therefore, must be reckoned as an important factor in the conversion of a superficial abrasion into a chronic sore.
- (b) Deficient blood-supply.—When the human stomach is artificially injected it at once becomes evident that the mucous membrane in the cardiac and central zones of the viscus receives the greater portion of the blood which enters the organ, for it

can be observed that the tissues in the pyloric region are often only partially injected when the rest of the stomach is fully engorged with the colouring material. This peculiarity in the vascular distribution is in strict accordance with the physiological law which ordains that the blood-supply of a tissue shall be proportionate to its functional activity, since it is the mucous membrane of the fundus and central portions of the organ which is mainly concerned with the processes of digestion and absorption.

But in addition to this there are two other conditions which adversely affect the circulation in the pyloric region. In the first place, the vessels supplying the mucous membrane at this spot pass obliquely through a considerable thickness of muscular tissue, and are consequently apt to suffer from compression at each contraction of the organ. The reality of this phenomenon is easily demonstrated by passing a strong electric current through the stomach of an animal, when the mucous surface of the pylorus is observed to become anæmic. In the second place, the arterioles that supply the pyloric region of the stomach are far more often attacked by atheroma than the other gastric vessels, and in most men who have passed the age of fifty some degree of thickening of the intima of the vessels in this part of the stomach can be detected with the microscope. Since the repair of any tissue depends to a great extent upon its supply of arterial blood, it is obvious that, other things being equal, a lesion situated near the pylorus will heal less rapidly than one which occupies a more vascular portion of the organ.

(c) Absence of submucous tissue.—It is worthy of notice that the mucous membrane in the pyloric region is firmly attached to the muscular coat instead of being separated from it, as in other parts of the organ, by a layer of loose connective tissue. This arrangement is obviously devised to prevent the prolapse of the mucous coat through the orifice during the contractions of the viscus, but it is distinctly inimical to the healing of an ulcer, as it not only prevents the contraction of the edges of the sore, but exposes them to the ever-varying strain which is produced by the movements of the muscular tissue.

(d) Adhesions.—It might be imagined that the formation of adhesions between the base of a pyloric ulcer and some solid organ in its neighbourhood, such as the liver or pancreas, would

be directly helpful to the process of repair by reducing the tissue to a state of comparative rest. Unfortunately, however, the principal effect of such adhesions is to prevent the base of the sore from undergoing the degree of contraction which is absolutely necessary to cicatrisation, in much the same way as the adhesion of an ulcer of the leg to the surface of the tibia so often retards cicatrisation.

(e) Spontaneous degeneration.—It has long been known that the mucous membrane in the pyloric region of the stomach habitually undergoes a form of fibroid degeneration after middle age. This phenomenon consists essentially of an overgrowth of the connective tissue between the tubular glands, whereby these structures become compressed, and finally obliterated. Occasionally the new tissue undergoes fatty degeneration. These changes are not associated with disease of the rest of the stomach, and are usually more pronounced in men than in women. When the fibrosis is extreme it is apt to be followed by chronic ulceration of the surface. Three instances of this kind are contained in the records of the London Hospital. In each it is distinctly stated that the diseased condition of the mucous membrane had preceded the ulceration.

2. Chronic Congestion

Embarrassment of the venous circulation through a tissue is almost as inimical to repair as a deficiency of arterial blood. This fact, which is obvious in a varicose ulcer of the leg, is exemplified in the case of the stomach by the frequency with which a chronic ulcer in the cardiac region is associated with diseased states of the heart (p. 90). It will be shown later that ulcers which are associated with congestion of the gastric tissues are usually characterised by repeated attacks of hematemesis.

3. Hyperacidity

An excessive acidity of the gastric juice not only tends to irritate an open sore, and thus to prevent it from healing, but by increasing the digestive activity of the secretion it helps to enlarge the size of the ulcer. It is now admitted that about 66 per cent. of all chronic ulcers of the stomach are accompanied by the condition of hyperacidity; and although

the origin of this abnormal condition is still open to discussion, there is no doubt that when once it has become established it offers a formidable obstacle to cicatrisation.

CONSTITUTIONAL CONDITIONS WHICH INTERFERE WITH REPAIR

The clinical fact that anamia frequently precedes the formation of a gastric ulcer suggests that an abnormal state of the blood may possibly prevent the repair of an abrasion of the mucous coat. This inference is strongly supported by the experiments of Daettwyler, who found that after dogs had been rendered anamic by repeated venesection, not only did slight sources of irritation give rise to ulceration, but that such ulcers were very slow in healing. Silbermann obtained similar results by the administration of chlorate of potassium and other drugs which produce hæmoglobinuria. It is also worthy of notice that the acute variety of gastric ulcer in young women is adversely influenced by severe hæmatemesis; the greater the loss of blood the longer the time that is required to ensure cicatrisation.

A deficiency of hæmoglobin may also be responsible for the slow repair of an ulcer in persons who suffer from malaria or tertiary syphilis; for it is well known that while such cases usually resist the ordinary methods of treatment, they often improve rapidly under the use of quinine, arsenic, or iodide of potassium.

Conclusions.—Although our knowledge concerning the causation of gastric ulcer is still very imperfect, the facts which have just been related appear to warrant us in coming to the following conclusions:—

An 'acute' ulcer of the stomach or duodenum may either appear as a primary affection or develop during the course of some organic or constitutional disease. These two varieties differ from one another in three important particulars. In the first place, the 'primary' form is usually solitary, though occasionally two or even three ulcers may be found in the same stomach, while the 'secondary' disease is invariably multiple, and affects the greater portion of the organ Secondly, the 'primary' affection is most frequently encountered in the

central or cardiac regions of the stomach in the immediate vicinity of the lesser curvature, while the 'secondary' variety is usually scattered over the whole of the inner surface, especially in the fundus and along the great curvature. Thirdly, the 'primary' ulcer is small in size, circular in outline, and penetrates so deeply into the coats of the viscus as frequently to open a large vessel or to perforate the entire wall. The 'secondary' ulcer, on the other hand, is very variable both in size and shape, and, though it occasionally involves the vessels of the submucous coat, seldom destroys the whole thickness of the organ.

Acute primary ulceration often commences as a hæmorrhage into the inner coats of the stomach during the hyperæmia which takes place at each menstrual epoch, and under other conditions. If the extravasation is small and superficial, the resultant erosion rapidly heals; but if the hæmorrhage is deep and extensive, or if the oxidising power of the blood is deficient, owing to a diminution in the quantity of hæmoglobin, repair will be delayed, and the erosion may be converted into a definite perforating ulcer. It is also possible that the disease may sometimes originate in a small infarct produced by the lodgment of a clump of disintegrated blood corpuscles in a capillary vessel.

Acute secondary ulceration may arise in many different ways, the order of their frequency being probably as follows: (1) Passive congestion of the stomach, such as occurs in cardiac and liver disease, leading to hæmorrhagic effusions into the gastric mucosa and subsequent erosion. (2) Severe catarrhal inflammation, which may be caused either by direct irritation by the ingesta, by septic absorption from burns of the skin, &c., or by the toxic products which accumulate in the blood in specific infectious fevers and disease of the kidneys. (3) Obstruction of an arteriole by the micro-organisms of pyæmia, glanders, anthrax, and other infectious diseases, or by emboli composed of pigment and disintegrated blood corpuscles, as in malaria and hæmoglobinuria. (4) Injury to the gastric tissues by severe blows upon the epigastrium, or by the swallowing of mineral acids and other corrosive fluids. (5) Inflammation of the solitary glands, which occasionally occurs spontaneously, but more often ensues during the course of some febrile affection, such as diphtheria, typhoid, or tuberculosis. (6) Thrombosis

of a vessel, either primary or consecutive to malignant disease of the organ.

Chronic ulcer of the stomach or duodenum usually commences in an insidious manner, but it is also apt to follow the acute disease in young women. The former mode of origin characterises the variety of the complaint which occurs in men about middle age. In these cases the ulcer is almost invariably situated in the pyloric region of the stomach, on the posterior surface, and nearer the upper than the lower border of the organ. It commences by the superficial digestion of a portion of the mucous membrane, whose vitality has been diminished by one or other of the following conditions: (1) The gradual obliteration of a nutrient vessel by atheromatous, fatty, lardaceous, or syphilitic disease. (2) Chronic inflammation of the mucous and submucous tissues. (3) Fibroid or fatty degeneration of the mucous membrane, such as occurs spontaneously after the age of forty. (4) Local anamia of the gastric wall from pressure of a tumour upon a vessel, compression by a fibrous band or calculous gall-bladder, or by the growth of a fibro-myoma in the wall of the viscus. In all cases the chronicity of the disease is favoured by the proximity of the ulcer to the movable pylorus, by the morphological peculiarities of the tissues, by passive congestion of the organ from failure of the heart or obstruction of the portal vein, and by concomitant hyperacidity.

When a chronic ulcer results from the acute disease, it is usually found in the middle or cardiac end of the stomach near the lesser curvature, and on the anterior surface. It exhibits a greater tendency to heal than the ordinary pyloric variety, and often owes its chronicity to neglect of treatment, concomitant anæmia, disease of the heart, or to some constitutional state such as syphilis, malaria, or phthisis.

PART II

CHAPTER I

SYMPTOMATOLOGY

It has hitherto been the custom of authors to describe the clinical features of a gastric ulcer as though the disease were invariably chronic in character, and never exhibited a sudden onset or pursued a rapid course. This failure to distinguish between what are essentially two distinct forms of the complaint has led to considerable confusion, since in the absence of a pathological basis of classification it has been found necessary to differentiate many clinical varieties of the complaint according to the exceptional prominence of some particular symptom. We therefore propose first of all to describe the general features of the acute varieties of the disease, and subsequently to deal with the ordinary chronic ulcer.

It has been shown that an acute ulcer may either occur as a primary disease, or develop in a more or less accidental manner during the course of some other disorder. There is thus a natural classification into primary and secondary acute ulceration of the stomach which is amply sufficient for all purposes of description.

1. Acute Primary Ulcer of the Stomach

This disease is sometimes called the 'acute perforating ulcer of young adults,' owing to its inordinate frequency between the ages of fifteen and twenty-five. Women are far more liable to it than men, the ratio of the two sexes, according

to our statisties, being nearly ten to one. The complaint is extremely eommon among domestic servants, governesses, dressmakers, and others in whom an indoor occupation is apt to induce a condition of anima or general ill-health.

The symptoms which attend the disease depend upon the situation of the uleer and the depth to which it penetrates. If, as is usually the ease, the uleer develops close to the lesser curvature, it is extremely apt to erode a branch of the eoronary artery. Consequently, vomiting of blood is one of the most frequent and notable features of the complaint. On the other hand, if it does not happen to involve a vessel, sudden perforation of the stomach may be the first symptom to attract attention. This is particularly apt to occur when the disease attacks the anterior wall of the viseus, where the larger arteries are some distance apart, and protective adhesions rarely form. Finally, if the ulcer neither crodes a blood-vessel nor perforates the peritoneum, the symptoms to which it gives rise are either quite unimportant, or they consist of pain after food, with occasional vomiting.

An analysis of 118 cases of acute primary uleer of the stomach which were treated at the London Hospital and London Temperance Hospital gives the following results:—

Severe hæmatemesis was the first symptom in eighty-nine cases, or 75.4 per cent.

Pain after food, with or without vomiting, was the earliest complaint in twenty-five eases, or 21.2 per eent.

Perforation of the stomach was the first symptom in four

cases, or 3.4 per eent.

These figures afford a fair idea of the relative frequency with which hamorrhage, pain, and perforation constitute the earliest indication of the disease.

(1) Hæmorrhage into the Stomach (Hæmatemesis and Melæna).—The bleeding from an acute uleer of the stomach is usually the result of the erosion of a medium-sized branch of the eoronary artery, and is therefore sudden and profuse. As a rule the capillary oozing which accompanies the destruction of the mucous coat is too insignificant to attract attention, although occasionally traces of altered blood may be observed in the vomit. The occurrence of the hæmorrhage is, of course, quite fortuitous, but the accident would seem to be exceptionally frequent during the period of gastric digestion or

after physical exercise. Thus out of fifty-seven cases where the patient was able to give a detailed account of the attack, in thirty-seven the hæmorrhage occurred within two hours of a full meal, and usually during the performance of some manual labour, such as scrubbing the floor, washing clothes, or arranging furniture; in twelve, when the stomach was presumably empty, and the patient engaged in some light occupation; and in the remaining eight, when the organ was empty and the body at rest. The influence of digestion upon the production of hæmorrhage may be explained by the distension of the organ, its vascular engorgement, and by the corrosive action of the gastric juice; while the injurious effects of physical exertion are probably due to the increased movements of the diaphragm exerting a strain upon the vessels of the lesser curvature.

The symptoms that arise from bleeding into the cavity of the stomach vary according to the quantity of blood which is As a rule, the rupture of the vessel is quite painless, but occasionally the patient experiences a sudden stabbing pain or trickling sensation in the epigastrium, or feels as though something had given way. Within a few minutes the loss of blood induces vertigo, faintness, or extreme weakness, which causes her to seek a recumbent posture. In some instances actual syncope occurs, while in young children a convulsion may be the first symptom of the accident. If the patient is asleep at the time, she usually awakens with a feeling of intense anxiety, and leaves her bed to seek assistance. The accumulation of blood in the stomach almost invariably excites nausea and vomiting, so that in a few minutes hæmatemesis occurs. In some cases, however, the blood appears first to stimulate the peristaltic movements of the intestine, so that the patient experiences griping pains in the abdomen, and an urgent call to stool, which is followed by hæmatemesis. In rare instances the blood is entirely eliminated by the bowel without any vomiting. This fact is one of extreme importance, and should always be borne in mind whenever a young female becomes suddenly anæmic without obvious cause. In the following case the neglect to examine the stools almost cost the patient her life.

Case I. A young lady who had always enjoyed excellent health was suddenly attacked with extreme weakness, fainting, and palpitation, which increased to such an extent that she could not be lifted

from the pillow without fainting. There was no complaint of pain, nausea, or vomiting, and the food was taken without difficulty or discomfort. No suspicion of the real nature of the case was entertained for several days, the treatment being directed to strengthen the action of the heart. When, however, the stools were inquired for and examined they were found to contain a large quantity of altered blood; and after the usual treatment was adopted to prevent a recurrence of the hamorrhage, the patient made a slow but perfect recovery.

Melæna without hæmatemesis is often regarded as indicative of duodenal as distinct from gastric ulceration, but this supposition is erroneous. The following case shows that even æsophageal hæmorrhage may give rise to melæna without any vomiting of blood.

Case II. A crossing-sweeper, fifty-two years of age, was admitted into the London Temperance Hospital on account of extreme debility and shortness of breath. The patient stated that he had been in good health until two days previously, when he had been attacked with faintness and had vomited a little blood. He had suffered from syphilis, and had led a very intemperate life. Examination showed that the face and lips were markedly anæmic, the pulse small and quick, and the temperature 99° F. The percussion note beneath the left elavicle was dull, and there was an entire absence of breath sounds as far down as the fourth rib. A distant thud could be detected on careful auscultation, and the left pulse was rather smaller than the right. There had never been pain in the back or any difficulty of swallowing. The liver was enlarged, the lower border of the stomach extended two inches below the navel, and there was some ascites. There was no epigastric tenderness, and no pain had ever been experienced after food. The condition of the urine indicated the existence of chronic interstitial nephritis. The stools were loose and contained a large quantity of altered blood. The diagnosis was 'aneurism of the thoracic aorta at the junction of its transverse and descending portions; eirrhosis of the liver; chronic gastritis, with probably secondary ulceration near the cardiac orifice; and chronic Bright's disease.'

Within the next few days the patient rapidly improved, and the blood disappeared from the stools. On the sixth day, however, he was again attacked with faintness, followed by diarrhea, and enormous quantities of blood were voided by the bowel; there was no vomiting or hiematemesis. All attempts to arrest the hamorrhage were futile, and he succumbed to failure of the heart on the following day. At the autopsy a large ancurism was found at the left bend

of the thoracic aorta, which was firmly adherent to the structures at the back of the chest, and had quite compressed the upper lobe of the lung. The sac was entirely filled with laminated clot, with the exception of a small cavity about the size of a walnut, which communicated by a minute hole with the æsophagus. The stomach and intestines were filled with the blood which had trickled down the æsophagus. The stomach itself was dilated, and its mucous membrane showed signs of chronic inflammation, but no ulceration could be found. The liver and kidneys were cirrhosed.

The quantity of blood which is vomited varies greatly in different cases, but is always considerable. As a rule the bleeding takes place rapidly, so that within a few minutes six to ten ounces are ejected, and this may recur several times in the course of the following hour. In other cases there is only one attack of hæmatemesis, and the whole quantity of blood, amounting to a pint or more, is vomited at once. When the initial leakage is very slight, two or three ounces of dark blood are first vomited; but as the rupture increases in size the effusion becomes more rapid, and a second attack results in the rejection of a large quantity of bright blood. It is obvious, therefore, that the chief features of the hæmatemesis of acute gastric ulcer are its suddenness, profuseness, and comparatively rapid cessation, thereby offering a marked contrast to the hæmorrhage from a chronic ulcer, which usually recurs several times, and may continue with brief intermissions until death takes place from exhaustion.

The reason why the hæmorrhage in acute ulcer usually ceases spontaneously is that the edges of the disease are soft and yielding and the vessels healthy—conditions which permit the eroded artery to contract sufficiently to close the aperture. In the chronic disease, on the other hand, the vessel lies like a rigid pipe in the fibrous wall of the ulcer, and, being unable to contract, can only become occluded by the process of clotting.

The appearance of the blood varies according to the rapidity with which it has been poured into the stomach. When the eroded vessel is small and the leakage comparatively slow the ejecta are usually liquid in consistence, dark red or 'prune juice' in colour, and mixed with small fibrinous coagula. As a rule, however, the bleeding is profuse, and emesis occurs before the gastric juice has time to act upon the hæmoglobin,

so that the blood is bright red, clotted, and alkaline in reaction. In rare cases it coagulates in the stomach and forms a cast of the organ. Thus, in one of our cases, a young girl, while apparently in perfect health, suddenly complained of faintness, and about half an hour afterwards was attacked with violent retching, as the result of which she vomited a solid cast of the stomach nearly four inches in length and one inch and a half in thickness. The mass was flexible, and was composed entirely of coagulated blood. With the exception of a small tear in the part which represented the lesser curvature, it was absolutely intact. The patient had no more hæmorrhage, and made a perfect recovery.

The general symptoms of gastric hæmorrhage are identical with those which follow a sudden loss of blood from any other organ of the body. Even before hæmatemesis has occurred the patient often becomes pallid and restless, and exhibits the inspiratory dyspnæa which has been aptly termed 'air-hunger.' These symptoms are often intensified by the anxiety engendered by the sight of the blood; indeed, we have known cases where moderate hæmatemesis occurred at night without the patient being aware that she had suffered from anything more serious than a bilious attack until she saw the blood on the following morning. Within a short time the surface of the body and extremities becomes cold and clammy, the pulse increases in frequency while it diminishes in volume, and a feeling of intense weakness, faintness, or vertigo is experienced. Occasionally palpitation of the heart, dimness of vision, noises in the ears, or a sense of emptiness and sinking at the epigastrium are notable features of the attack; or the patient may suffer from actual syncope. Dryness of the mouth and thirst arc invariably present. If the loss of blood is excessive, complete collapse may occur. During the continuance of the hæmorrhage the pulse is quick, small, and compressible, and in bad cases may cease to be felt at the wrist, while the temperature of the body is markedly depressed.

As soon as the bleeding has ceased, reaction sets in, and the pulse increases in volume, though it still continues to exhibit the compressible and jerky character of an incompletely filled artery. In most instances the temperature also recovers itself, and may rise one or two degrees above the normal. This febrile reaction varies with the severity of the hæmorrhage and the previous condition of the patient, being most marked where the general health before the accident was good, and the bleeding moderate in amount.

During this period the cheeks become slightly flushed, the eyes sunken and surrounded by dark lines, the lips dry and cracked, while sordes may collect about the teeth. Owing in a great measure to the prohibition of solid food the tongue becomes dry and coated with a grey or brown fur, while the palate and throat are apt to be attacked by thrush. Thirst is always a prominent feature of the case, but all desire for food

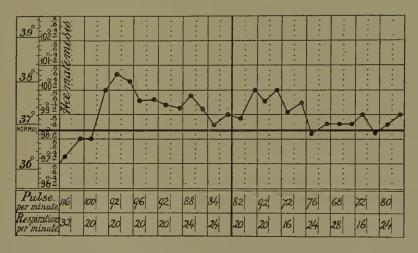


Fig. 37.—Showing the degree of pyrexia which usually follows an attack of hæmatemesis (anæmic fever).

is usually lost. Among the other symptoms of this condition, throbbing in the head, noises in the ears, palpitation, insomnia, and a feeling of uncontrollable restlessness are most frequently the subjects of complaint.

As a rule there are no gastric symptoms whatever, and even when severe pain has preceded the hæmatemesis it almost invariably subsides after the hæmorrhage has taken place. Localised pressure upon the epigastrium often gives rise to pain or to a feeling of sickness. The bowels are confined, and it may not be until after they have been opened once or twice that a black appearance of the stool proves that some blood has found its way into the intestines. In other cases the first evacuation is found to contain blood, and several liquid tarry motions may be passed in rapid succession.

The amount of blood which is voided in this way is usually in inverse proportion to the quantity vomited. Quantitative examination of the corpuscles and hæmoglobin of the blood in the circulation is always difficult to perform on account of the depletion of the superficial vessels, but in several cases where an estimation was made we found that the red corpuscles did not exceed 50 per cent. of the normal. The greater the degree of depletion the longer is the time required for the adequate renewal of the blood, more especially if the patient has suffered from anæmia previous to the hæmorrhage. In these latter cases severe anæmia may continue for many months, or even years, despite the wholesale administration of iron. In most instances, however, it disappears within three months.

(2) Pain and Vomiting.—Pain after food constitutes the initial symptom of the disease in about 21 per cent. of the cases of acute primary ulcer of the stomach.

This symptom varies considerably in character, for we find that out of our twenty-five cases where the complaint commenced with discomfort after meals, in nine, or 36 per cent., severe pain was complained of, while in the remaining sixteen, or 64 per cent., the patient likened the sensation to that of 'ordinary indigestion.' It is obvious, therefore, that only about 7 per cent. of the cases of this dangerous disease complain of that severe form of pain which is supposed to be characteristic of gastric ulcer.

When actual pain occurs it is caused by direct contact of the food with the ulcerated surface, and with the surrounding mucous membrane, which is swollen and hyperesthetic. In these cases the disease is usually situated in the pyloric region and on the posterior surface of the organ. As a rule the symptom first makes its appearance from ten to twenty minutes after a meal, and is referred to the epigastrium rather than to the chest. It is chiefly excited by the ingestion of solids, and often subsides immediately the patient is restricted to a milk diet. Unless vomiting occurs it may last for an hour or more, or may continue the greater part of the day. An attack of emcsis always relieves, though it may not entirely remove it. In rare instances the pain is continuous, and bears no relation to food, but is increased by physical exercise. These cases are particularly dangerous, on account of the hæmatemesis or perforation of the stomach which often ensues.

When the gastric symptoms merely resemble those of atonic dyspepsia, the ulcer is usually situated at the upper border of the stomach near the cardia or upon the anterior surface. Almost immediately after a meal, or even when only a few mouthfuls of food have been swallowed, the patient experiences a sense of weight and discomfort in the chest and between the shoulders, accompanied by distension of the abdomen and flatulence. It is important to observe that these phenomena are quite as readily provoked by the ingestion of fluids as solids, and are aggravated rather than relieved by a milk diet. It is probable that in such cases the stomach is always moderately distended with gas, the tension of which is suddenly raised by the introduction of food. Nearly all the patients are anæmic and have previously suffered from indigestion, so that they usually regard their symptoms as due to a recurrence or an exacerbation of their former complaint. Occasionally there is a burning sensation in the left hypochondrium and beneath the left breast, while the skin in these regions feels bruised and is very tender upon pressure.

Although nausea and waterbrash are frequently present, actual vomiting is comparatively rare, and when it does occur it usually fails to relieve the pain to the same degree as in cases of chronic ulcer. Constipation is almost invariable. The tongue is red, clean, and pointed when the pain is severe, but pale, flabby, and indented along its margins by the teeth when flatulent dyspepsia is the prominent symptom.

The urine is pale in colour, neutral or faintly acid in reaction, and deposits phosphates on standing. Coldness of the extremities, a variable appetite, disinclination to physical and mental exertion and a dry, lank state of the hair are minor symptoms of which complaint is sometimes made.

(3) Perforation of the Stomach.—In rather more than 3 per cent. of our cases sudden perforation of the stomach constituted the first symptom of the disease. As a rule, however, this accident is preceded for several days by severe pain in the epigastrium, which is aggravated by food or exercise. It is comparatively rare after hæmatemesis, on account of the careful treatment to which persons who have vomited blood are always subjected.

The initial symptoms of perforation are practically the same in all cases, but the subsequent phenomena vary according

to the degree of peritonitis which is excited. If, as is usually the case, perforation takes place soon after a meal and involves the anterior surface of the stomach, a considerable quantity of the acid contents of the organ finds its way into the general cavity of the peritoneum, and acute general peritonitis ensues. On the other hand, it occasionally happens that the ulcer is situated at the upper border and perforates the peritoneum at a time when the stomach is empty, so that only a few drops of fluid or bubbles of gas escape from the viscus. Under these conditions the resultant peritonitis is slight, and is strictly localised at the site of the injury.

From the notes of ninety-eight cases of gastric perforation by an acute ulcer we find that the first symptom in every instance was a sudden, agonising pain in the abdomen, which was occasionally accompanied by a sensation of something giving way in the belly or of an internal gush of fluid. As a rule the pain was first referred to the epigastrium, but in a few instances it seems to have been located in the hypogastric or iliac regions. In 16 per cent. of the cases severe collapse ensued immediately afterwards, while in 8 per cent. an attack of syncope was followed by prolonged unconsciousness. Vomiting as an early symptom of perforation appears to be comparatively rare, as it was only recorded in about 29 per cent. Its infrequency is attributed by Traube to the rapid escape of the gastric contents through the artificial aperture, but it is more probable that reflex emesis is inhibited by the profound shock which rapidly supervenes.

From the very first the condition of the patient is obviously one of extreme gravity. The face rapidly acquires the pallid, anxious, and pinched expression which characterises most acute diseases of the abdomen, and the patient often expresses her conviction that the attack will be fatal. The pulse is rapid, small, and feeble, and, when the collapse is severe, may be imperceptible at the wrist. The respiratory movements are quick and shallow, and principally costal in character. The surface of the body is cold and of a dusky blue colour, while the head and extremities are bathed in a cold, clammy sweat. The breath feels chilly, and the internal temperature is reduced

several degrees below the normal.

As a rule the patient lies flat upon the back, with the knees drawn up and the head low. Consciousness is usually retained,

but the mental condition is apathetic, and the voice slow, feeble, or husky. The bowels are confined. Extreme thirst is always present. In many cases the secretion of urine is suppressed for several hours, while in others the desire to micturate is frequent and accompanied by straining and pain. The urine may contain both albumen and casts. Blomfield has observed in men retraction of one testicle similar to that which occurs in renal colic. During the first few hours after the accident the abdomen is usually hard and retracted, owing to the spasmodic contraction of the superficial muscles, and the slightest pressure upon the epigastric and umbilical regions gives rise to great pain.

If a considerable quantity of gas escapes from the stomach it usually finds its way between the anterior surface of the liver and the diaphragm, and causes the percussion note over this region to become tympanitic instead of dull. This physical sign is only of value in the early stages of the complaint, as when peritonitis has set in the intestines become distended with gas, and push the liver upwards and backwards so that the hepatic dulness is usually obliterated. It is very rare for the gastric contents to accumulate in the peritoneal cavity in such quantity as to give rise to the signs of free fluid in the abdomen.

The stage of collapse usually lasts from six to ten hours, and gradually disappears as the symptoms of peritonitis manifest themselves. Death at this period occurred in about 4 per cent. of our cases, and was due either to sudden paralysis of the heart or to gradual failure of the respiratory centre. In the former the patient drops down as though she had been shot, while in the latter the fatal termination is ushered in by profound coma. Crisp relates the case of a girl fifteen years of age, who, when apparently in perfect health, suddenly uttered a scream and fell down insensible. She died on the following day without recovering consciousness. At the autopsy the brain was found to be quite healthy, but a small ulcer of the stomach had perforated into the peritoneal cavity. The following case was somewhat similar in its nature.

Case III. A young lady who had never suffered from any gastric trouble suddenly complained of pain in the abdomen, giddiness, and diarrhœa. She went to bed, and no anxiety was felt about her, but

when visited shortly afterwards she was found to be comatose. An autopsy showed that the stomach had been perforated by an acute ulcer. The other organs were healthy.

In about 96 per cent. of the cases the perforation of an acute ulcer is followed by general peritonitis, while in the other 4 per cent. the inflammation remains localised to the site of puncture. The greater frequency with which local peritonitis ensues from the perforation of a *chronic* ulcer is due to the protective adhesions which have formed round the base of the latter disease before the actual rupture takes place.

Acute General Peritonitis.—General suppurative peritonitis is almost certain to take place under the following circumstances: (1) when perforation occurs soon after a full meal, and is followed by severe collapse which persists for more than four hours; (2) if within a few hours of the accident there are pain and tenderness over the whole of the abdomen, with suppression of urine or difficulty of micturition; (3) if the liver dulness disappears immediately after the accident, or free fluid (gastric contents) can be detected in the peritoneal cavity.

The first symptoms of peritonitis usually show themselves from eight to twelve hours after the onset of the pain. Although the surface of the body may continue cold, the internal temperature rises, and may soon attain a point several degrees above the normal. The pulse also increases in rapidity and becomes small and wiry. The general appearance of the patient indicates the increasing gravity of her condition. The eyes appear to retreat into the orbital cavities, the face looks blue and pinched, and the expression is one of acute pain and anxiety.

The respiratory movements are quick, superficial, and entirely costal in type; and to aid the breathing the patient often holds the top of the bedstead, or lies with the hands behind the head. The mouth is dry, and great thirst is complained of. Occasionally retching and hiccough are troublesome symptoms. The abdomen is uniformly distended, tympanitic on percussion, and exquisitely tender on pressure.

Micturition is difficult and painful, and absolute retention is not infrequent. The bowels are confined. As the disease progresses these various symptoms are intensified. The pulse increases in frequency, but diminishes in volume, and eventually

becomes extremely feeble and irregular; the inspiratory efforts are jerky in type and followed by prolonged, noisy expirations; the mind is slightly clouded, the voice whispering, and the hiccough incessant. The pulsations of the heart are feeble and irregular, the bases of the lungs congested, and sometimes free fluid may be detected in the peritoneal cavity. As a general rule death occurs from cardiac failure from eighteen to thirty-six hours after the onset of the pain, but occasionally life is prolonged for three or four days. We know of an instance where a girl left her bed on the third day and undertook a long railway



Fig. 38.—Four-hour chart in a case of general peritonitis from perforation of a gastric ulcer. The accident occurred about three hours before admission into the hospital.

journey, although suffering at the time from general purulent peritonitis. Where the patient is supposed to have lived for a week or more (nineteen days: Jackson), it is probable that the peritonitis was circumscribed at first and afterwards became diffused.

Localised Peritonitis. The signs which indicate that the peritoneal inflammation will probably remain circumscribed are the converse of those which point to the supervention of diffuse peritonitis, namely, rapid recovery from the initial shock, absence of diffuse tenderness over the abdomen and of difficult micturition, and persistence of the liver dulness. In almost every instance there is evidence to show that the stomach was empty at the time of the accident. In such cases the pulse begins to regain its volume within two or three hours, the extremities become warm, and the anxious, pinched

expression of the face gradually disappears. The pain also diminishes, and may only be felt upon deep inspiration or coughing. Although the breathing is quick and shallow, the diaphragm can be seen to move slightly with respiration. The mind remains clear, and, as the collapse passes off, the patient is inclined to regard her condition in a hopeful light. The temperature of the body may show a slight reaction, but it seldom rises above the normal, and usually remains depressed for a week or two. Secondary fever is never observed unless a perigastric abscess forms, or the bowels are allowed to remain confined. Constipation is an invariable symptom, and epigastric pain is apt to follow an evacuation. Nausea, flatulence, pain between the shoulders, and inability to sleep are frequent sources of complaint for the first few days. Examination of the abdomen shows it to be slightly distended, more especially in the epigastrium. The superficial muscles are rigid, and pain is experienced when pressure is made over the region of the stomach. After the lapse of four or five days the distension diminishes, and it is then sometimes possible to detect a localised thickening in the epigastrium with an impaired note on light percussion. This condition is due to the presence of inflammatory exudation round the site of the puncture, with adhesion of the omentum, and it may endure for several months. After recovery has taken place the patient usually suffers from weak digestion and troublesome constipation. The following case, which has recently come under our care, is a good example of this variety of perforation of the stomach from acute ulcer.

Case IV. An unmarried lady, 27 years of age, who had suffered from pain after food for ten days, was attacked, when getting out of bed one morning, with sudden violent pain in the upper part of the abdomen, which caused her to utter a scream and fall fainting upon the floor. When help arrived she was still unconscious, and did not revive for some time. She then complained of great pain in the region of the stomach, and a feeling of impending dissolution. The extremities were cold, the pulse hardly perceptible, and the temperature in the rectum 96° F. There was no vomiting.

Three hours later, when the collapse had partially disappeared, the patient stated that at the age of 24 shc had a sudden attack of hæmatcmesis, but since that time she had never been troubled with discomfort after meals until the commencement of the present

illness. The bowels had been regular and the general health good.

The face was very pale, the eyes sunken, and the nose pinched and cold. The pulse was feeble and slow (68), the respirations costal in type, and the urine scanty, but passed without difficulty or pain. The upper part of the abdomen was slightly distended and its walls rigid. Considerable pain was experienced when the body was moved or pressure made over the epigastrium or left hypochondrium. Below the level of the umbilicus the abdomen was flaccid and free from tenderness. The diaphragm moved slightly on deep inspiration. On the following day the patient appeared better, but still complained of much pain in the epigastrium. The temperature remained about 97°, the pulse 68, and the respirations 22. The tongue was clean, but dry along the centre. The whole of the

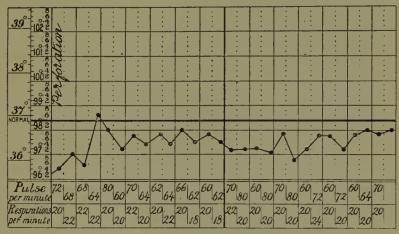


Fig. 39.—Temperature chart in a case of perforation of the stomach with spontaneous recovery.

epigastric region was swollen, and very tender upon pressure, the percussion note being tynipanitic. The base of the left lung was resonant on percussion and the vesicular murmur normal. On the third day the pain had diminished, but there was constant belching of wind and an aching sensation between the shoulders. The temperature had risen to about 100° F. during the night, and the pulse rate increased to 96, but these symptoms soon subsided. On the fifth day pain was only experienced upon coughing or moving the body, and the bowels had responded freely to an enema of castor oil. The epigastric region was less distended, and on careful palpation an indistinct tumour could be detected a little to the left of the median line, which was extremely tender, and afforded a dull note on light percussion. The tumour became more distinct on the eighth day, after which time it gradually disappeared. In the meantime the

patient rapidly improved, and within a short time epigastric pain was only felt when the bowels were opened. At the end of a month she was allowed to sit up daily, and she soon regained her strength. The only troubles at the present time are constipation and some pain in the region of the stomach when the bowels act. This condition is probably due to adhesion of the great omentum to the stomach and the transverse colon.

Acute Secondary Ulcer of the Stomach

There are three principal conditions in which secondary ulceration of the stomach is attended by symptoms of clinical importance, viz. (a) in pyæmia and other diseases where a general infection occurs; (b) in cases of portal obstruction with congestion of the stomach; (c) after direct injury to the organ.

A. INFECTIVE DISORDERS

The multiple ulcers which are sometimes found in the stomach after death from pyæmia, erysipelas, and the various specific fevers are usually regarded as devoid of any clinical interest. That this complication, however, is far more frequent than is supposed, and is often the immediate cause of death, is evident from the following cases, which we have excerpted from the records of the London Hospital and London Temperance Hospital.

Case V. Pyæmia: acute ulceration of stomach. A woman, 75 years of age, was admitted into the hospital for a compound fracture of the bones of the left forearm. The wound was suppurating, and the patient suffered from repeated rigors and other symptoms of pyæmia. There was no history of vomiting or pain after food.

Autopsy. Both pleuræ were coated with recent lymph, and the left chest contained a small quantity of pus. The liver presented several abscesses. The mucous membrane of the stomach was intensely inflamed, and two recent ulcers, each about the size of a shilling, were found in the pyloric region of the organ. The edges of the sores were sharply defined, and their bases extended almost to the peritoneum.

Case VI. Pyæmia: acute ulceration of stomach: fatal hæmorrhage. A man, 38 years of age, was attacked by pyæmia after sustaining a compound fracture of the left ulna. For three days before his death he vomited small quantities of altered blood, but never complained

of any pain. He was suddenly seized with faintness and dyspnœa, became unconscious, and died within half an hour.

Autopsy. The stomach and intestines were filled with blood. Several ulcers of recent formation, varying in size from a sixpence to a shilling, were present on either side of the lesser curvature of the stomach. One of these ulcers had almost perforated the wall of the organ, its base being formed by the peritoneum, while another



Fig. 40.—Acute ulceration of the stomach in pyaemia. Photograph; natural size. (London Hospital Museum.)

had exposed and eroded a large branch of the coronary artery. A photograph of the stomach is shown in fig. 40.

Case VII. Pyamia: acute ulceration of stomach: fatal hamorrhage. A middle-aged man was admitted into the hospital for hydatid of the liver accompanied by jaundice. The cyst was aspirated, and thirty ounces of fluid were withdrawn.

About a week later the temperature began to rise, and the patient suffered from repeated rigors and attacks of retching. On the twelfth day the epigastrium was slightly distended and tender, and the ejecta contained altered blood. There was no pain after food, but retching was incessant. Death occurred suddenly from failure of the heart.

Autopsy. General peritonitis. The liver contained two cysts, one of which was filled with clear fluid and the other with pus. The stomach was distended, and contained nearly a pint of blood. The eardiac region of the organ was studded with numerous recent ulcers, which varied from the size of a split pea to that of a sixpence. Some were quite superficial, while others had exposed the peritoneum. The fatal hæmorrhage was due to erosion of a branch of the eoronary artery.

Case VIII. Erysipelas: acute ulceration of stomach: fatal hæmorrhage. A man, about 37 years of age, who had led a very intemperate life, was attacked by erysipelas after amputation of the arm. On the third day he complained of great pain at the pit of the stomach, and retched incessantly until his death, which occurred two days later. No mention is made of blood in the ejecta.

Autopsy. The stomach was filled with pitchy fluid. The whole surface of the gastrie mucous membrane was profusely studded with hæmorrhages, many of which had been converted into superficial ulcers. The bleeding appeared to have taken place from several ulcers, as no large vessel was found to be eroded.

Case IX. Anthrax: acute ulceration of the stomach. A woolsorter was admitted into the hospital with severe bronchial and abdominal symptoms. There were much pain and tenderness of the epigastrium, with vomiting and diarrhea.

Autopsy. The mucous membrane of the stomach was found to be extremely injected and profusely studded with hæmorrhages interspersed with circular perforating uleers. Some of these were small and superficial, but the majority were of considerable size, and had deeply penetrated the coats of the organ. The blood in the vessels of the stomach contained the characteristic bacilli.

Case X. Enterie fever: acute ulceration of stomach: fatal hæmatemesis. A girl, thirteen years old, was admitted into the hospital with the symptoms of enteric fever of eight days' duration. Vomiting occurred on three occasions, but there was no complaint of pain. At the end of the fourth week, when the temperature had begun to decline, the patient was seized with profuse hæmatemesis, from which she never rallied.

Autopsy. The anterior wall of the stomach was found to be adherent to the under surface of the liver. Scattered over the whole surface of the organ were numerous uleers with sharply defined edges, the largest of them being about the size of a florin. The adhesion to the liver was caused by local peritonitis over the base of an uleer which had almost perforated. The eroded vessel was not found. The first part of the duodenum contained a large ulcer of recent formation.

Case XI. Enteric fever: acute ulceration of stomach. A young

woman succumbed to typhoid in the third week of the disease, without having exhibited any gastric symptoms during the course of her illness.

Antopsy. Three circular perforating ulcers were found in the pyloric region of the stomach near the lesser curvature. A photograph of this specimen is shown in fig. 41.

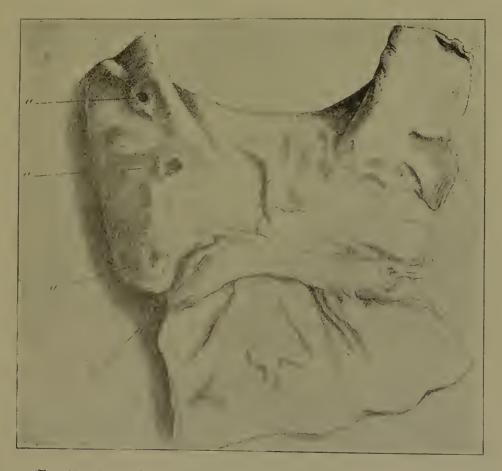


Fig. 41.—Acute ulcers (a, a, a) in the stomach in a case of enteric fever. (London Hospital Museum.)

Case XII. Acute pneumonia: acute ulceration of the stomach: fatal hæmorrhage. A boy, ten years old, was admitted into the hospital for acute inflammation of the right lung. Death occurred rather suddenly on the fifth day of the disease. There had been no vomiting or hæmatemesis.

Autopsy. The stomach was filled with elotted blood. On the lesser eurvature, one inch from the pylorus, were two round ulcers, separated from one another by a thin bridge of mueous membrane. The larger measured one inch, and the smaller rather more than half an inch, in diameter. Both ulcers presented sharply defined

edges, and had penetrated to the peritoneum. On the floor of the

larger one was an eroded branch of the coronary artery.

Case XIII. Varicella followed by noma. Acute ulceration of the stomach. A female child, twenty months old, died in the hospital from noma following varicella. After death two recent ulcers were found on the anterior wall of the pyloric region of the stomach, close to the lesser curvature.

Case XIV. General tuberculosis: acute ulceration of the stomach. An infant, twenty-two months old, died in the hospital from general tuberculosis. During the last week of life retching and vomiting were incessant, but there was no hæmatemesis.

Autopsy. The whole of the inner surface of the stomach was profusely studded with small superficial, circular ulcers, the largest of which was about a third of an inch in diameter. The solitary glands were much enlarged, but there were no signs of tubercle.

Case XV. General tuberculosis: acute ulceration of the stomach. A little girl, about two years of age, was admitted into the hospital for tubercular meningitis, from which she died on the following

day.

Autopsy. Several recent ulcers were found in the stomach, the largest of which was situated upon the anterior wall of the fundus, and measured half an inch across. It had penetrated to the peritoneum. The other ulcers were somewhat smaller in size and occupied the central and pyloric portions of the viscus. There were no signs of tubercle in the stomach, although the peritoneum was studded with grey granulations.

With regard to the symptoms exhibited by these different cases, it is to be observed that pain after food and vomiting were seldom present. On the other hand, hæmorrhage, due to the erosion of a large vessel, was the immediate cause of death in no fewer than five out of the eleven cases. In one instance (case x.) the patient succumbed after an attack of hæmatemesis, but in the other four a sudden failure of the heart was supposed to be due to the effects of the primary disease until the autopsy showed that the stomach and intestines were full of blood. These facts are of considerable importance, for they not only indicate that fatal hæmorrhage from the stomach may occur during the course of a specific infective disease, but that the accident is seldom attended by hæmatemesis. Although actual perforation of the stomach never took place, it was recorded several times that one or more of the ulcers had penetrated as far as the peritoneum, while in one instance (case vi.)

it appeared as though perforation must have taken place had not life been suddenly cut short by hæmorrhage. Leith has recorded an instance of gastric ulcer following pneumonia in a child, in which perforation gave rise to a local abscess, which eventually burst through the diaphragm.

B. CONGESTION OF THE STOMACH

Obstruction to the venous circulation through the stomach is apt to give rise to superficial hæmorrhages in the mucous coat at the cardiac end of the organ, which subsequently develop into small ulcers (p. 91). This morbid condition is particularly common in disease of the mitral valve, but it is also met with in cirrhosis and other diseases of the liver which cause congestion of the portal system. Owing to the small size and superficial character of the ulcers the symptoms to which they give rise are indistinguishable from those that accompany the coexisting state of gastric congestion. Actual pain is seldom complained of, though much discomfort may ensue from flatulent distension of the stomach. Vomiting is almost invariable, and takes place immediately after food, the ejecta being mixed with mucus and bile or with traces of altered blood. Occasionally retching is a constant and troublesome feature of the case. The appetite is completely lost, the tongue is coated with a creamy fur, and the breath has the sweet odour of acetone. Slight jaundice is often present. The epigastrium is tender, owing to congestion of the liver, the left lobe of which overlaps the greater portion of the stomach.

Although small in size, the ulcers are very apt to erode a dilated vein or a branch of the coronary artery, and to cause severe and even fatal hæmorrhage; but perforation rarely occurs. These facts are well illustrated in the following cases.

Case XVI. Mitral stenosis: acute ulceration of the stomach: fatal hæmatemesis. A woman, 35 years of age, was admitted into the hospital for dropsy of the abdomen and extremities due to stenosis of the mitral valve. For more than a week she had vomited everything she had taken, and on several occasions the ejecta had been streaked with blood. There were marked cyanosis of the face and slight jaundice. The urine was scanty and albuminous. Three days later she was attacked by profuse hæmatemesis which recurred at short intervals and rapidly brought life to an end.

Autopsy. Marked stenosis of the mitral valve with great dilatation of the left auricle and right chambers of the heart. Congestion and ædema of both lungs with moderate serous exudation in the pleural cavities. Nutmeg liver, and old infarcts in the spleen and kidneys. Stomach and intestines contained a large quantity of blood. Numerous small crosions scattered over the inner surface of the stomach and upper part of the duodenum. Close to the cardiac orifice of the stomach on the lesser curvature were two circular ulcers, the larger of which was about the size of a three-penny piece. The base was covered with blood-clot, and when this was removed an eroded branch of the coronary artery was exposed.

Case XVII. Mitral stenosis: acute ulceration of the stomach: fatal hæmatemesis. A man, 28 years of age, was admitted into the hospital for vomiting of blood. He had always been a temperate man, and had never suffered from any gastric complaint. On examination the mitral valve was found to be stenosed and incompetent, the liver was slightly enlarged, and the ankles ædematous. The patient was quite blanched, and died soon after admission from cardiac failure.

Autopsy. Mitral valve thickened and incompetent. Much blood in the stomach and intestines, and many punctiform hæmorrhages in the fundus. About one inch to the inner side of the cardiac orifice, and close to the lesser curvature, was an ulcer the size of a hempseed, which had penetrated into a large branch of the eoronary artery.

Case XVIII. Mitral disease: acute ulceration of stomach: hæmatemesis: death from pneumonia. A man aged 47 years was admitted into the hospital for dilatation of the heart. Examination showed that the mitral valve was incompetent. Enlargement of the liver was present, with ascites and ædema of the legs. The urgent symptoms had existed for about three weeks. Soon after admission the patient brought up a considerable quantity of blood during an attack of retching, the bleeding being repeated a few hours later. After two days acute pneumonia set in and rapidly proved fatal.

Autopsy. Stenosis and incompetence of the mitral valve. Acute pneumonia of the right side. Granular contracted kidneys. In the centre of the stomach near the lesser curvature was a small oval ulcer, and on the inner side of the cardiac orifice were two circular ulcers each about the size of a sixpence. The edges of the sores were sharply defined and devoid of thickening. On the floor of one of them was an eroded branch of the coronary artery with an adherent clot.

Case XIX. Mitral disease: aeute ulceration of stomach: fatal hæmatemesis. A woman about thirty years of age was visiting a

friend who was a patient in the hospital, when she suddenly felt faint, and vomited a large quantity of blood. She was removed to bed in a state of collapse, and died soon afterwards.

Autopsy. Incompetence of the mitral valve with thickening of the aortic cusps. The stomach was filled with blood, and close to the cardiac orifice on the lesser curvature was a circular ulcer about



Fig. 42.—Photograph of a stomach, showing a minute ulcer near the cardiac orifice, which gave rise to fatal hæmorrhage. A glass rod has been passed through the branch of the coronary artery which was croded. (London Hospital Museum.)

one-eighth of an inch in diameter which had perforated a large branch of the coronary artery. This specimen is shown in fig. 42.

Case XX. Aertic disease: acute ulceration of stomach: futal hæmatemesis. A woman, 42 years of age, was admitted for dilatation of the heart. On examination both sides of the organ were found to be much enlarged, and the aertic valves incompetent. There were the usual signs of back pressure, accompanied by vomiting of all food and medicine. Soon after admission the patient brought up about half a pint of bright blood, became collapsed, and died within an hour.

Autopsy. Incompetency of aortic valves. Nutmeg liver, with a large calculus in the gall-bladder. Stomach and intestines full of blood. At the cardiac end of the lesser curvature was a small superficial ulcer, on the floor of which a large vein could be seen with a ragged hole in its wall.

Case XXI. Cirrhosis of liver: acute ulceration of stomach: fatal hamatemesis. A man, aged 40 years, who had led a very



Fig. 43.—Varicose veins in the stomach, with acute ulceration. Death from hæmorrhage. Photograph; natural size. (Museum of the Royal College of Surgeons.)

intemperate life, was admitted into the hospital for ascites due to cirrhosis of the liver. After the lapse of a month he was seized with violent hæmatemesis which continued until death took place a few hours later.

Autopsy. Much ascites. Liver markedly contracted and cirrhosed. Granular kidneys. Hypertrophy of the left ventricle. Much blood in stomach and intestines. At the cardiac orifice was a minute superficial ulcer which had opened a large varicose vein that lay immediately beneath it.

Case XXII. Cirrhosis of liver: acute ulceration of stomach: fatal hæmatemesis. A middle-aged man was brought into the hospital in a dying condition, having vomited a large quantity of blood in the street.

Autopsy. A small quantity of fluid in the peritoneal cavity. Well-marked cirrhosis of the liver. Stomach and intes-

tines contained much blood, but no ulcer was observed by the pathologist. Subsequently when the veins of the stomach were injected, the fluid was observed to escape from a pore-like ulcer concealed among the folds of mucous membrane near the cardiac orifice. The vein from which the hæmorrhage had taken place was greatly dilated.

During the same period of time in which the foregoing cases occurred, eleven other instances of acute gastric ulceration, associated with portal congestion, were observed in the post-mortem room of the London Hospital. It would therefore seem that severe hæmorrhage occurs in about one-third of the fatal cases of gastric ulcer due to this cause. It is also interesting to notice that whereas in cases of cardiac disease the bleeding usually ensues from crosion of a branch of the

coronary artery, the ulcer which develops in cirrhosis of the liver is particularly apt to lay open a dilated vein.

C. TRAUMATIC ULCER

Acute ulceration of the stomach may follow a blow on the abdomen or back if the injury has been sufficiently severe to give rise to hæmorrhage into the submucous coat of the organ. Cases of this description have been recorded by Lebert, Brinton, Hoggan, Tapié, and other writers. The first symptom of the accident is usually an attack of hæmatemesis, which may recur several times, and is followed by pain and vomiting after food, and the other symptoms indicative of gastric ulcer. Perforation, however, is very rare, as the disease ceases to progress as soon as the necrotic tissue has been removed. Healing usually occurs within two or three months, but occasionally the ulcer may assume a chronic character (Potain). The following cases, which have come under our own observation, will serve to illustrate the principal features of the complaint.

Case XXIII. A labourer, 42 years of age, who had previously enjoyed excellent health, was engaged soon after his midday meal in turning a windlass, when the handle slipped and, revolving backwards, struck him in the abdomen with great force. The blow gave rise to considerable pain, and the man was obliged to lie on the ground for about ten minutes before attempting to resume work. Half an hour later he began to feel faint and sick, and suddenly vomited a large quantity of bright blood. On admission into the London Temperance Hospital the patient was found to be in a state of eollapse, the face being blanched the extremities cold, and the pulse hardly perceptible. There was a small bruise two inches above and slightly to the left of the navel, but there was no fluid in the peritoneal eavity. Soon after admission he vomited about eight ounces of clotted blood, and a somewhat smaller quantity was rejected a few hours later. When the collapse had passed off, the bowels were moved, but there was no blood in the evacuation. Tablespoonful doses of iced milk did not occasion either nausea or pain. On the third day the patient complained of an aching pain at the epigastrium, and twice vomited after taking a small quantity of milk. The superficial bruise was about the size of a florin, but considerable tenderness was present over the whole of the epigastrium. As the result of an enema a large quantity of black blood was eliminated by the bowel, and the stools continued to exhibit signs of altered blood for three days. The patient continued

to complain of pain in the abdomen, in the region of the bruise, which for the first ten days was always increased by the ingestion of milk and other fluids, so that the nutrition was chiefly maintained by means of nutrient enemata. Subsequently this symptom diminished, and at the end of a fortnight six ounces of milk could be taken every two or three hours without discomfort. In the third week a trial was made of some boiled fish, but this had to be discontinued on account of the pain which it excited. In the sixth week the patient was able to eat lightly boiled eggs, thin bread and butter, and raw meat pulp without pain or discomfort, and from this time onward he improved so rapidly that he was discharged cured eight weeks after the accident. The stomach was then found to be normal in size and free from tenderness on pressure. No free hydrochloric acid could be detected after a test meal. Two days after the accident the temperature rose to 99.4° F., and continued to range between 99° and 100° for four days, after which it fell to normal. When the patient was seen six months after the accident he stated that his general health was perfect, and that he was quite free from any discomfort after food.

Case XXIV. A man, about 22 years of age, who had been previously free from any gastric disturbance, received a violent blow upon the back from a cricket ball. The following day he was attacked by hæmatemesis, which recurred two or three times within the next few days, and was followed by the usual symptoms of gastric ulcer. The pain after food was very severe and accompanied by vomiting, and the complaint resisted treatment for several months.

Foreign bodies introduced into the stomach seldom give rise to acute ulceration of the organ, although cases have been recorded where iron nails, glass, hardware, and even a straw have produced perforation of the organ through the medium of ulceration (Marks, Sanford). The ulcer which ensues from the ingestion of corrosive poisons, although acute at first, almost invariably pursues a chronic course, and will therefore be alluded to in Part III. Ulceration of the stomach from pressure applied to the epigastrium is well illustrated by the famous case recorded by Murchison, in which a hysterical female actually produced a gastric fistula by means of a coin.

Acute Ulceration of the Duodenum

Like its homologue in the stomach, an acute ulcer of the duodenum may either occur as an idiopathic or primary complaint, or ensue during the course of some other malady.

1. Acute primary ulcer of the duodenum is a rare disease. It is usually met with between the ages of 18 and 30, and is far more common in men than in women. In six fatal cases observed at the London Hospital five were males, the average age at the time of death being 24 years. Owing to the small size of the ulcer and the fact that the food is reduced to a kind of pulp before it reaches the duodenum, pain or discomfort after meals is rarely sufficiently pronounced to attract the attention of the patient. The only signs, therefore, which can be said to indicate the presence of the disease are sudden melæna, with or without hæmatemesis, and perforation of the bowel. The former accident is rarely fatal, owing to the healthy condition of the vessels and the absence of induration in the wall of the ulcer. The only case with which we are acquainted was particularly interesting, as the fatal hæmorrhage occurred at a time when the ulcer had almost healed.

Case XXV. A man, 25 years of age, was admitted into the London Temperance Hospital for vomiting of blood. There was no history of any gastric trouble, and the various organs of the body appeared to be free from disease. The bowels were opened two or three times after admission, and on each occasion a considerable amount of dark blood was evacuated. The following morning the patient brought up six ounces of bright blood, and passed more than a pint of darker blood into the bed. He became blanched and breathless, and died somewhat suddenly.

At the autopsy, the intestines were found to contain much blood. About one inch from the pylorus, on the outer wall of the duodenum, there was a small flat cicatrix of recent formation, in the centre of which an eroded branch of the pancreatico-duodenal artery was exposed. The other organs were normal.

It may be said, therefore, that whenever a young man in apparently good health is attacked by melæna, with or without hæmatemesis, it is probable that the ulcer is located in the first part of the duodenum rather than in the stomach, the converse being the case in young women.

Unfortunately, owing to the sparseness of large vessels upon the anterior surface of the duodenum, and the comparative thinness of its wall, sudden perforation of the bowel is usually the first symptom. This occurred in every one of our six cases of the disease, the cause of death being general peritonitis. The symptoms of the accident are identical with those which accompany perforation of the stomach (page 142).

2. Acute secondary ulceration of the duodenum occasionally occurs in cases of portal congestion or during the course of the various specific fevers, but is most frequently encountered in pyæmia and septicæmia. The form which is apt to follow burns of the skin is the only one that possesses any clinical importance.

In these cases the disease probably commences about the third day and attains its maximum development in the second week. Curling found that one-half of his cases suffered from abdominal pain, and about one-third from vomiting, but subsequent investigations seem to show that these initial symptoms are usually absent.

An attack of diarrhœa sometimes precedes the onset of the characteristic phenomena of the complaint, which, as in other forms of intestinal ulceration, consist of hæmorrhage and perforation. Melæna, with or without hæmatemesis, occurs in about 30 per cent. of the fatal cases. The bleeding almost invariably results from the erosion of a large branch of the pancreatico-duodenal artery, and may either give rise to immediate death or so exhaust the patient that he falls a prey to pneumonia or to some other intercurrent affection. A small quantity of blood in the dejecta is by no means uncommon during the first week after a severe burn, but this is due to the intense congestion of the intestinal mucous membrane (Erichsen). Perforation is rare, and has never been observed at the London Hospital, though Curling states that the ulcer may progress so rapidly as to expose the pancreas. The natural tendency of the disease is to cicatrisation; and from the fact that scars are often found in the duodenum in persons who have formerly suffered from burns, it is probable that in many instances the ulceration heals without any untoward symptoms.

CHAPTER II

DIAGNOSIS AND PROGNOSIS

It is easy to diagnose an acute ulcer of the stomach when some characteristic symptom such as hæmatemesis or perforation is present, but if the disease is merely accompanied by pain or discomfort after food its recognition is usually difficult, and

may be impossible.

Vomiting of blood occurs from other causes besides gastric ulcer, so that the mere statement of the patient that she has brought up blood cannot be accepted off-hand as an indication of the disease. Many anæmic girls suffer from retching in the early morning, as the result of which a little mucus tinged with blood derived from the throat may be ejected. Sometimes disease of the naso-pharynx gives rise to slight hæmorrhage, and if this occurs during the night the blood may be swallowed unconsciously and vomited on the following morning. Still more common is the bleeding that ensues from diseased teeth or spongy gums; in one case, which was sent to us on account of repeated hæmatemesis, the source of the blood was discovered in a malignant growth of the tonsil, the existence of which had been overlooked. Infants occasionally vomit blood which they have sucked from a crack in the mother's nipple. while those who have been brought up entirely upon sterilised milk and starchy foods are apt to suffer from a scorbutic condition of the gums that gives rise to an oozing of blood, which is swallowed by the child and afterwards vomited. It must never be forgotten that hysterical women and malingerers will often inflict superficial injuries upon the tongue or mouth in order to simulate gastric hemorrhage, and will sometimes swallow the blood of animals for the same purpose. Finally, a colour somewhat similar to that of altered blood may be produced in the vomit by the presence of the salts of iron and bismuth, by red wines and coffee, by the juice of cranberries, and by bile.

In all these cases, however, the quantity of blood is small, and the spurious hæmatemesis is frequently repeated, whereas in cases of acute ulcer the hæmorrhage is always profuse, and when once it has ceased it does not recur except in considerable quantity. It is only necessary therefore to consider those conditions in which a large amount of blood is usually rejected.

In every case the first point to be determined is whether the blood has come from the stomach or from the lung. This question would be easily settled if the patient could state with certainty whether she had coughed up the blood or vomited it; but as a matter of fact the two efforts are so often combined that usually no certain clue can be obtained from the history of the case.

The distinction between hæmatemesis and hæmoptysis has to be made by directing attention to the following points: (1) An attack of hæmatemesis is usually preceded by a feeling of faintness, nausea, and giddiness, while hæmoptysis often begins with palpitation of the heart and tickling in the throat. (2) If hæmatemesis occurs after a meal, the blood is often dark, mixed with food and slightly acid in reaction, whereas that derived from the lung is bright red, frothy, and alkaline. (3) Hæmatemesis is arrested as soon as the blood has ceased to flow into the stomach, but in cases of hæmoptysis the patient continues to spit up dark nummular clots for several days after the more copious hæmorrhage has terminated. (4) Hæmatemesis is usually followed by the appearance of black blood in the stools, but this only occurs in very severe cases of hæmoptysis where the patient has been obliged. to swallow the blood to prevent himself from choking. (5) Hæmatemesis either takes place suddenly when the patient is apparently in good health (acute ulcer) or after he has suffered for some time from pain and vomiting, but hemoptysis is usually preceded for many weeks by pain in the chest, cough, loss of flesh, and night sweats. (6) In the former complaint the physical signs are slight tenderness upon pressure over the epigastrium, the stomach can be often felt distended with blood, and there is usually increased pulsation in the abdominal aorta; whilst in the latter, dulness on percussion, crepitation, or some other indication of pulmonary disease can generally be detected on examination of the chest.

Table 13. - Differential Diagnosis of H.ematemesis and H.emoptysis

	Hæmatemesis	Hæmoptysis		
1. Onset	Preceded by nausea, faint- ness, or vertigo	Preceded by palpitation, tickling in the throat, or cough		
2. Appearance of blood	Bright red and clotted if stomach is empty, but dark, acid, and mixed with food if hæmorrhage oc- curs after a meal	Bright red, frothy, and alkaline		
3. Sequelæ	Melæna	Expectoration of dark num- mular clots for some days		
4. Previous history	Usually pain after food and vomiting	Usually cough, pain in the chest, loss of flesh, and night sweats		
5. Physical signs.	Occasionally localised ten- derness in epigastrium; often distension of the stomach and increased pul- sation of abdominal aorta	Dulness on percussion of chest; tubular breathing, or crepitation		

Before the age of thirty the principal causes of profuse hæmatemesis are acute gastric ulcer, chronic gastric ulcer, duodenal ulcer, and cirrhosis of the liver. In acute gastric ulcer the hæmorrhage is generally the first symptom of the complaint, and the patient is almost invariably a young woman; while in the chronic form of the disease pain after food and vomiting have usually been experienced for a considerable time. and the patient is often of middle age. It is also to be observed that the hæmorrhage of acute ulcer ceases rapidly, and rarely returns after a day or two; while that of chronic ulcer may recur several days in succession, and also at subsequent periods. Duodenal ulcer is more common in men than in women, and the quantity of blood passed by the bowel greatly exceeds that which is eliminated in the vomit. If pain is present it occurs two or three hours after food. When hæmatemesis ensues from cirrhosis of the liver there is a history of intemperance, with discomfort after meals, nausea and retching in the early morning, or bleeding piles. The liver may be enlarged, or ascites may be present.

Hæmatemesis is said sometimes to take the place of the catamenia, and cases of amenorrhæa have been recorded where a regular recurrence of the gastric hæmorrhage appeared to warrant this belief (Watson). It must be observed, however, that in certain cases of chronic ulcer of the stomach hæmat-

emesis only occurs at the menstrual periods, and under these conditions the catamenia are often suppressed. It is therefore possible that some of the recorded instances of vicarious menstruation were of this nature. Finally, it has been affirmed that hæmatemesis presents itself in hysteria, but we are not aware of any case where a careful examination of the stomach after death has disproved the existence of an ulcer or of a ruptured vein. When hæmatemesis ensues after the cure of piles the patient is usually suffering from cirrhosis of the liver, while its occasional alternation with epistaxis, intestinal hæmorrhage, or hæmaturia may be explained by the existence of hæmophilia or some other blood disease.

When pain after food constitutes the principal symptom, acute ulcer of the stomach has to be distinguished from two other complaints which are extremely common in young and anæmic women. The first of these is usually termed 'gastric hyperæsthesia,' though our personal opinion is that the symptoms are more often due to the constant presence of gas in the stomach than to any increase of sensibility of the mucous membrane, while the second is essentially an intestinal neurosis, a violent and painful contraction of the colon being provoked in a reflex manner by the introduction of food into the stomach. A differential diagnosis can usually be made by attention to the following points:—

(1) In gastric hyperæsthesia the pain ensues immediately after food, and is referred to the left hypochondrium as well as to the epigastrium, while in ulcer it does not usually develop until after an interval of ten to twenty minutes, and is chiefly experienced in the epigastrium. (2) In the former complaint pain follows the ingestion of liquids as well as solids, but in the latter it usually subsides as soon as the patient is restricted to a milk diet. (3) Vomiting may occur in both diseases, but in ulcer it usually appears at the crisis of the painful attack, the symptoms of which it alleviates, while in gastric hyperæsthesia it often takes place immediately after food, and only affords partial relief. (4) In ulcer there is usually localised tenderness over the epigastrium, but in gastric hyperæsthesia the whole of this region is tender, while the skin over the left hypochondrium and beneath the left breast is extremely sensitive. (5) The symptoms of ulcer are readily allayed by rest in bed, a milk diet, and a course of

bismuth and morphine, but those which depend upon gastric hyperæsthesia require the administration of iron. It must never be forgotten that acute ulceration of the stomach is often associated with a hyperæsthetic state of the surrounding mucous membrane, so that in all cases where the symptoms are severe the patient should be treated as though she were suffering from the more serious complaint.

The chief characteristics of the colonic disorder are: (1) The pain which follows the ingestion of food resembles that of ordinary colic, being referred to the umbilical region, and appearing to cross the abdomen from right to left. (2) In severe cases it may give rise to vomiting, but as a rule this symptom is absent. (3) During the attack there is often an urgent call to stool, though an actual evacuation seldom occurs. (4) There is usually tenderness on pressure along the course of the transverse and descending colon, but not over the epigastrium. (5) Constipation is almost invariable, though attacks of diarrhœa due to colonic catarrh are apt to supervene from time to time. (6) The disorder is readily cured by a course of suitable laxatives and iron.

The symptoms of perforation of the stomach have chiefly to be distinguished from those which accompany severe colic, appendicitis, and pelvic hæmatocele.

In cases of colic there is often a history of previous attacks of a similar kind or of lead poisoning. The pain comes on gradually, increases in severity until it reaches its maximum, and afterwards subsides. Vomiting and syncope may occur at its climax, but collapse, like that which ensues from gastric perforation, is rarely observed. The abdomen is retracted, but pressure relieves rather than increases the pain. The liver dulness persists, there is no intestinal distension, and an action of the bowels affords immediate relief.

Appendicitis is more common in men than women. There is often a history of a previous attack, or of antecedent constipation with discomfort in the region of the cæcum. The pain is first referred to the right iliac fossa, and subsequently becomes diffused over the abdomen. Collapse is often absent, and is never so severe as in perforation of the stomach. There is tenderness over the cæcum and over 'McBurney's spot,' along with resistance on palpation, and sometimes a tumour can be felt per rectum or in the iliac fossa.

In pelvic hæmatoeele there is sudden abdominal pain, anæmia, and collapse, but these symptoms are much less pronounced than in gastric perforation, while the pain is usually referred to the hypogastrium. General tenderness of the abdomen, with loss of liver dulness, is never observed. Vaginal examination shows that the uterus is displaced owing to a swelling situated posteriorly or at one side.

Table 14.—Differential Diagnosis of Perforation from Acute Gastric Ulcer

Disease	Previous history	Pain	Collapse	Physical signs
1. Perforated gastric ulcer.	Often pain after food for some days	Onset sud- den, vio- lent, epi- gastric	Immediate, severe, persistent	Abdomen rigid and tender; absence of liver dulness; cos- tal respiration
2. Colic .	Former at- tacks or indi- gestible meal	Onset gra- dual, grip- ing, um- bilical	Very rare	Abdomen retracted; pain relieved by pressure
3. Appendicitis .	Former at- tack, or con- stipation and pain in right groin	Sudden, severe, in right iliac region	Moderate or absent	Usually vomiting; maximum tender- ness over cæcum with resistance on palpation; always some fever
4. Pelvic hæmato-cele.	Irregular menstrua- tion or over- exertion	Sudden, moderate, hypogas- tric	Moderate or absent	Sudden anæmia; vomiting; tender- ness over hypogas- trium; uterus dis- placed; a swelling in Douglas's pouch or in fornix

Examination of the contents of the stomach does not afford any clue to the existence of an acute ulcer, nor is the employment of a tube justifiable in any case where there is a possibility of it being present. In one instance, where the patient was admitted into the hospital for 'acute dyspepsia,' the total acidity after a test meal was found to be 52, while the filtrate contained only a trace of free hydrochloric acid. A few days afterwards the diagnosis was changed to that of 'acute ulcer' on account of a severe attack of hæmatemesis; but the case is of interest as showing that the statements which have been made concerning an increase of the acid prior to the occurrence of hæmorrhage do not hold good with reference to the acute form of the complaint. Out of nincteen cases where the gastric contents were examined

one month after the hæmatemesis, free hydrochloric acid was absent in seventeen, while in the other two only a trace could be discovered; and this condition usually persisted until the patient had entirely recovered from the loss of blood. These observations are of some importance in view of the troublesome dyspepsia which so often follows severe hæmatemesis in early life.

The diagnosis of secondary ulceration of the stomach can be made only when there is persistent vomiting, accompanied by the rejection of small quantities of altered blood, or when severe hæmatemesis supervenes during the course of some constitutional or organic disease.

Prognosis.—The hæmatemesis which occurs from acute primary ulcer of the stomach is hardly ever fatal, for among our eighty-nine cases where this symptom was present death never ensued directly from loss of blood. Unless, however, the case is properly treated, there is always a danger of exhaustion from insufficient nutrition, while occasionally life is cut short by an attack of pneumonia or some other intercurrent disease.

Formerly, perforation of the stomach followed by peritonitis was invariably fatal, but the modern treatment of this complication by immediate abdominal section has greatly reduced its mortality.

Persons who have once suffered from the disease are very liable to another attack, and it is interesting to observe that the second ulcer is extremely apt to terminate in perforation. This is partly due to the frequency with which it attacks the anterior surface of the stomach, and partly to neglect of its early symptoms, which are often indistinguishable from those of the coexisting dyspepsia. Our statistics upon this point are necessarily imperfect, but we find that nearly 20 per cent. of our cases were readmitted into hospital within three years for a recrudescence of their former symptoms.

There are three sequelæ of acute ulceration which merit some degree of attention, namely, chronic ulceration, persistent dyspepsia, and severe anæmia. Chronic ulceration is very common in cases which, not having suffered from hæmatemesis, have not been subjected to proper medical treatment. In such the disease is usually situated in the middle of the stomach near the lesser curvature, and may remain in an indolent state for many months. Owing to the slow

progress of the ulcer, local peritonitis is often set up about its base, so that when perforation takes place the results of the accident are strictly circumscribed, and a perigastric abscess is produced (Part IV. Chapter 5).

Persistent dyspepsia is most common after a severe attack of hæmatemesis. The pain is experienced soon after food, and is referred to the chest and the dorsal spine. It is usually associated with flatulence, water-brash, and constipation, and is probably due to a deficiency of hydrochloric acid in the secretion.

Severe anæmia as a sequela of acute ulcer is comparatively rare, but we have seen several cases where a patient did not recover her colour for many months, or even years, after an attack of profuse hæmatemesis. This condition is quite distinct from the pernicious anæmia which is said to occasionally ensue from chronic ulcer of the stomach.

Acute duodenal ulceration is much more dangerous than the gastric variety, owing to its greater proclivity to perforation. Death from hæmorrhage is very rare.

The prognosis in cases of secondary ulcer of the stomach and duodenum usually depends upon the severity of the primary disease, but whenever hæmorrhage occurs it always increases the danger to life, and is not infrequently the immediate cause of death.

CHAPTER III

TREATMENT OF ACUTE GASTRIC ULCER

General Measures.—Rest in bed is essential to the treatment of all acute diseases of the stomach, and in no case are its beneficial results more pronounced than in acute ulceration. If hæmatemesis has occurred the patient should be confined to bed for three weeks or a month, but when dyspepsia is the principal feature of the complaint this period may be curtailed. During the stage of convalescence pressure upon the abdomen by corsets, bandages, or belts should be avoided.

Pain in the epigastrium rarely requires any special treatment, but should it prove troublesome a couple of leeches may be applied over the tender spot, or the upper part of the abdomen covered with a large linseed poultice. When the complaint occurs in a neurotic subject a small blister or the application of the liniment of iodine to the affected part is of considerable value.

Dietetic.—The principal point to be observed in the selection of a diet is to avoid all source of irritation and to give the stomach as much rest as possible. Theoretically this can be achieved by prohibiting food by the mouth, and maintaining the nutrition entirely by enemata; but, as a matter of fact, such a course is seldom necessary except during the first twenty-four hours after the hæmorrhage, while a long continuance of feeding solely by the rectum is often fraught with danger. All that is necessary is to provide a form of nourishment which does not irritate the surface of the ulcer nor unduly excite the secretory and motor functions of the stomach; requirements which can only be fulfilled by the use of a liquid diet.

Of all the fluid forms of nourishment which we have at our disposal milk is by far the most valuable, since it not only contains all the elements essential to nutrition, but also presents them in a form most easy of digestion and absorption.

As a rule fresh milk can be taken without discomfort, but occasionally it gives rise to oppression at the chest, nausea, flatulence, or acidity. Under these circumstances it should either be skimmed or mixed with a small quantity of lime water, or slightly alkalised with bicarbonate of sodium. Sterilised milk can often be borne when fresh or diluted milk disagrees, but we have known a scorbutic condition of the gums ensue from its long-continued administration. Peptonised milk is very valuable, especially when the powers of digestion have been impaired by hæmorrhage. If there is much pain after food, the milk should be cooled before use by means of ice, but as a rule it is most agreeable when slightly warm. It should be given every two hours, in quantities not exceeding six ounces at a time, the amount being gradually increased until the patient can take about three pints in the course of the twenty-four hours. The object of giving small doses at a time is to avoid distension of the stomach, which might lead to perforation of the ulcer or to rupture of a vessel.

Various meat juices and solutions have been recommended, but they are all inferior to milk. Leube's-beef solution is nutritious and non-irritant, but it is difficult to prepare in an ordinary household, and is often distasteful to the patient. Milk thickened with Benger's food, peptonised gruel, freshly expressed beef-juice, Liebig's extract of beef, and various meat jellies and essences may be employed to relieve the monotony of the milk

diet.

As it often requires a month for an acute ulcer to heal, at least four weeks should be allowed to elapse before an attempt is made to give solid food. By the adoption of this rule the chance of the ulcer assuming a chronic character is rendered remote. In all cases the addition of each new article to the dietary must be watched with care, and, if pain is experienced after it, recourse should be had once more to milk. In most cases, at the end of the month, bread and milk, powdered rice, arrowroot or sago puddings made with milk, and scrambled eggs may be allowed, followed at the end of another week by boiled chicken or cod which has been passed through a sieve, or raw meat pulp. Subsequently sweetbread, tripe, calves' brains and feet, boiled fish, poached eggs, and thin bread and butter may be given. At the end of the second month the patient can usually resume her ordinary

diet. Alcohol is seldom necessary, but if dyspepsia is troublesome during convalescence a small quantity of whisky or brandy in hot water given at the end of the meals often affords relief.

Medicinal.—As there are no drugs which exercise a specific curative effect upon the ulcer, the only indications for medicinal treatment are the relief of pain and the regulation of the bowels. It is only in rare cases that rest in bed, combined with a fluid diet, fails to remove the epigastric pain; but when these measures do not suffice recourse must be had to carbonate of bismuth, combined with bicarbonate of sodium. If vomiting is a marked feature of the case, it may be necessary to add a few drops of the solution of morphine or dilute hydrocyanic acid to the mixture, which should always be administered about ten minutes before food. Excessive flatulence may be relieved by the exhibition of carbolic acid or resorcine.

In all cases the bowels require careful regulation. This should be effected by means of enemata of soap and water, castor oil, or glycerine; but no purgative should be given by the mouth for the first month, to avoid increasing the peristaltic movements of the stomach and intestine. When the patient is able to leave her bed a small dose of cascara at night or an aloes and iron pill after meals is usually sufficient to secure a daily evacuation.

After-treatment.—As soon as the patient is able to take solid food an attempt should be made to improve the condition of the blood by means of iron. In some cases the solution of the perchloride answers very well, but as a rule the milder preparations, such as the ammonio-citrate, tartrate, or carbonate, are borne better. The drug should be given after the morning and midday meals, and should the tongue become white or the patient complain of nausea, the medicine should be omitted for a time in favour of a mixture containing rhubarb and bicarbonate of sodium, with an occasional dose of mercurial pill at night. It sometimes happens that a patient cannot tolerate iron in any form, and in such cases dioxide of manganese (grs. 2) forms a useful substitute.

The best treatment for the dyspepsia which so often follows acute ulcer of the stomach is to administer hydrochloric acid immediately after food, either alone or combined with iron, while the diet is strictly regulated, meat, soups, and farina-

ceous substances being excluded. A residence in a bracing locality is an extremely valuable adjunct to the treatment.

Treatment of Hæmatemesis.—In all cases the patient must be confined to bed.

It is usually taught by those who make no distinction between the acute and chronic forms of the disease that whenever hemorrhage occurs no food should be permitted for ten days or a fortnight, the general nutrition being sustained by nutrient enemata. This practice is often of great value in cases of chronic ulcer, where the eroded vessel has become plugged by a soft thrombus, which any functional excitement of the stomach might easily displace or dissolve; but it is seldom necessary in the acute variety of the disease, and when injudiciously employed it is fraught with considerable danger.

It was stated in the chapter upon 'Prognosis' that death occasionally ensues from exhaustion after hæmatemesis, but it might have been said with equal truth that the fatal issue is often due to starvation. Practitioners are very apt to forget that a person cannot be nourished indefinitely by the rectum, and that the rapidity with which absorption takes place from the bowel varies in different cases; consequently they often fail to realise that a patient may die from starvation despite the regular administration of nutrient enemata. The fact that many of the so-called cases of 'fatal hematemesis' in young adults succumb from want of nourishment rather than from loss of blood has repeatedly been brought home to us, both in hospital and private practice, and we believe that if this fact were more generally appreciated the loss of life from acute ulcer of the stomach would be greatly diminished. In all cases the condition of the pulse constitutes the best index of the state of the nutrition, a steady increase of rapidity combined with loss of tension being a certain sign that nourishment by the mouth is urgently required.

Case XXVI. A lady, 18 years of age, was seized with violent hæmatemesis after suffering from 'indigestion' for about a fortnight. She was put to bed, and nutrient enemata were administered every four hours, the bowel being cleansed night and morning. After the first attack there was no recurrence of the hæmorrhage. On the fourth day, however, she complained of great weakness, giddiness, and noises in the ears, and gradually became so exhausted that the doctor feared that internal hæmorrhage was taking place, and suggested a consultation.

When we saw her on the seventh day she was extremely feeble and slightly delirious. The lips and tongue were dry, the extremities cold, and the pulse 140 per minute. The abdomen was retracted, and no tenderness or tumour could be detected. The bases of the lungs were clear, and there was a loud hæmic bruit over the pulmonary and mitral areas of the heart. The nutrient enemata were stated to have been retained, and there had been no signs of blood in the evacuations since the fourth day. The temperature in the mouth was 100° F.

The general aspect of the case, combined with the absence of the usual signs of internal hæmorrhage, led us to believe that the rapidly increasing exhaustion was due to lack of sufficient nourishment, and we therefore recommended the administration of four ounces of peptonised milk by the mouth every hour, with eight ounces of peptonised gruel containing half an ounce of brandy by the rectum every eight hours. Within a few hours the patient's condition underwent a marked improvement; the pulse rate diminished, the delirium disappeared, and within a short time she was out of danger.

The safest plan is to give the stomach absolute rest for thirty-six to forty-eight hours, after which time, if the hæmor-rhage has not recurred, two to four ounces of peptonised milk may be allowed by the mouth every two hours, and a nutrient enema administered twice or thrice a day. On the fifth day rectal alimentation may be discontinued and the dose of milk increased to six ounces, while on the seventh day the usual diet suitable to the disease may be prescribed (p. 167).

There are several points of importance to be borne in mind with regard to rectal feeding. In the first place, a certain amount of time is always required for the absorption of an enema, so that at least three hours should be allowed to elapse between the injections. Secondly, however carefully the enemata are prepared, they almost invariably produce irritation of the rectum, to prevent which it is necessary to wash out the bowel at least twice in the twenty-four hours with a quart of warm water in which a teaspoonful of common salt has been dissolved. It may also be necessary to combine a few drops of laudanum with each nutrient enema. Thirdly, in order that they should be absorbed as quickly as possible, the injections themselves must be composed of materials which are easily absorbed, whilst they must not exceed a certain size lest they excite the peristaltic movements of the gut and act as aperients. With regard to this last point it was formerly the custom to

limit the size of a nutrient enema to two or three ounces, but of late years it has been shown that the employment of larger injections (eight to ten ounces) at longer intervals is quite as effective and less disturbing to the patient.

Of the various nutrient enemata the following are the most valuable: (1) Peptonised milk or beef tea, 10 ozs.; the yolk of 1 egg; peptone powder, 2 drachms; bicarbonate of sodium, 5 grains: every 6 hours. (2) Peptonised milk, 8 ozs.; the yolks of 2 eggs; a dessertspoonful of brandy, whisky, or rum: every 6 or 8 hours. (3) Warm milk or beef tea, 2 ozs.; the yolk of 1 egg; a teaspoonful of rum; tincture of opium, 5 mins.: every 3 hours. (4) Eight to ten ozs. of Leube's pancreatic meat emulsion 1 or of Roberts's peptonised milk gruel,2 or of freshly defibrinated ox blood: every 6 hours. Some practitioners add lactose to the enema, with the view of increasing its nutritive properties, while others employ prepared peptone for a similar purpose; but their value is doubtful. According to our experience no reliance can be placed upon nutrient suppositories.³ In those rare cases where extreme irritability of the rectum exists, Menzel and Porco, Whittaker, and others have advised the hypodermic administration of peptonised milk, olive oil, or beef essence, with inunctions of cod-liver oil; but although several cases treated in this manner have recovered, the method cannot be recommended as a successful substitute for the ordinary modes of feeding.

In the great majority of cases, when the patient is treated in the manner indicated, the hæmorrhage ceases spontaneously and does not recur, so that there is no obvious indication for medicinal treatment. If, however, there is much restlessness, a small dose of morphine may be injected with advantage. All purgatives must be avoided, and the bowels moved solely by means of enemata.

It sometimes happens that the warning conveyed by the first

¹ Leube's pancreatic emulsion is prepared as follows: Mix 6 ozs. of scraped and finely chopped beef with 2 ozs. of finely minced pig's pancreas, and rub them up in a mortar with a little water until the whole acquires the consistency of gruel. The syringe must have a wide nozzle.

² To prepare peptonised gruel, mix equal quantities of well-boiled gruel and fresh milk, and add to the enema two drachms of Liquor Pancreaticus (Benger) and five grains of bicarbonate of sodium.

The 'meat enules' lately introduced by Burroughs, Wellcome, & Co. are free from many of the objections that pertain to suppositories.

attack of hæmatemesis is neglected, and the patient continues to pursue her ordinary avocations and to place no restriction upon her diet. Under these circumstances the bleeding not infrequently recurs, and sometimes proves so excessive that special measures have to be adopted to restrain it.

In all cases of severe hæmorrhage the patient should be placed upon her back, with the head low, and the body covered with a sheet or light blanket, while an ice-bag is applied over the epigastrium with the view of controlling the movements

of the stomach.

The older writers placed the greatest reliance on the administration of styptics. Thus Fox speaks highly of acetate of lead in doses of three or four grains, combined with a quarter of a grain of opium, every three hours; while Brinton preferred ten grains of gallic acid, with ten minims of dilute sulphuric acid in one ounce of water. Large doses of perchloride of iron have also been advocated, but they are apt to excite vomiting. Turpentine was recommended by Hunter and Graves, and is of undoubted use in many cases. One or two teaspoonfuls of the oil in the form of an emulsion may be given at once, and followed after an hour by a further dose of thirty minims. When the bleeding is continuous but slight in amount, ten grains of gallic acid combined with a quarter of a grain of the extract of opium in the form of a pill, or five grains of the compound kino powder given every three hours, is often of great service. Alum whey and haseline are strongly recommended by some authorities, while others pin their faith upon hypodermic injections of ergotine (grs. 2-5). Calcium chloride is sometimes of value in chronic cases, but it must be given in full doses (grs. 30) every two hours.

Various local measures have been suggested to restrain hæmorrhage from a gastric vessel. In one of our cases where the patient appeared to be at the point of death, the stomach was irrigated with ice-cold water, with the result that the bleeding ceased and did not recur.

Schilling has proposed to tampon the stomach by means of a rubber bag attached to the end of a stomach tube, which can be inflated with air. In one case where this procedure was adopted the bleeding ceased within twelve minutes, and was not renewed.

Whenever it is possible, an attempt should be made to apply

direct pressure upon the upper part of the epigastrium by means of an abdominal tourniquet.

The ancient device of restraining internal hæmorrhage by the application of elastic ligatures to the extremities is occasionally useful, and should never be neglected in dangerous cases. The ligatures are placed round the upper parts of the arms and thighs, and are made just tight enough to compress the veins without interfering with the arteries. As the result of this a large quantity of blood accumulates in the limbs, and the internal organs are rendered proportionately anæmic.

If syncope threatens, brandy or ether may be administered subcutaneously, or an enema containing some diffusible stimulant may be given. It is unwise, however, to stimulate the heart to vigorous action, lest a sudden increase of arterial tension should displace the blood-clot which may have formed at the bleeding point.

When life is threatened from loss of blood recourse should be had to transfusion. For this purpose a saline solution (sod. chloride 6 parts, distilled water 1,000 parts) is to be preferred to defibrinated blood.

It must be remembered, however, that the sudden injection of a large quantity of fluid into the circulation is extremely apt to dislodge the protective clot and to cause a renewal of the bleeding. The cases recorded by Hacker, Légroux, and Roussel terminated fatally in this manner. A safer plan is either to transfuse small quantities (6–10 ozs.) at intervals of two or three hours, or to inject the saline solution into the cellular tissue of the axillæ, and at the same time to give a warm enema of the same composition. In a case which was recently under our care at the London Temperance Hospital these measures proved successful after the patient was apparently moribund.

The first suggestion of surgical interference in cases of hæmorrhage was made by Rydygier (in 1882), who advised that the ulcer should be cut down upon and excised. Although this idea was scouted at the time as being extravagant, it has since been successfully carried out by Mikulicz, Roux, and others. In two instances of uncontrollable hæmorrhage Küster opened the stomach and cauterised the base of the ulcer, and afterwards performed gastroenterostomy. Attempts have also been made to control the bleeding by means of a purse-string suture round the ulcer.

Undoubtedly the chief obstacle to the success of surgical interference is the exhausted condition of the patient at the time when the operation is undertaken. This of course is due to the fact that it is impossible to foretell whether medicinal treatment will control the hæmorrhage or not; and since the bleeding sometimes ceases when the patient is at death's door, the physician naturally hopes to the last that such will be the case. It is possible that at some future date abdominal surgery will prove as successful in the treatment of hæmorrhage as it is in perforation of the stomach.

When hæmatemesis occurs from secondary ulceration of the stomach in cases of heart or liver disease, a different line of treatment must be adopted. The principal indication is to relieve the congestion of the portal system, and with this object half a dozen leeches should be applied over the liver, while the bowels are freely moved by a dose of calomel followed by a saline draught. As soon as the gastric symptoms have abated, attention must be directed to the treatment of the primary

disease.

Treatment of Perforation.—In every case the patient must be placed as soon as possible in the recumbent position, and all movements of the body strictly prohibited. To relieve the pain, a pillow may be folded beneath the knees and a cradle put over the abdomen to remove the pressure of the bed-clothes. No food should be allowed by the mouth, as it is ant to find its way into the peritoneal cavity through the perforation. If stimulants are necessary they must be administered by the rectum or injected subcutaneously. To relieve the thirst, small pieces of ice may be sucked, or half a pint of hot water given in the form of an enema. Where there is much pain hot fomentations or turpentine stupes should be applied to the abdomen, and a small dose of morphine be given by hypodermic injection. When there exists any doubt as to the diagnosis, sedatives must be given sparingly so as not to mask the symptoms; but under other circumstances opium or morphine should be exhibited to control the movements of the bowels, and to counteract as far as possible the effects of shock.

Surgical Treatment.—Until the year 1880 the treatment of this serious complication consisted in those medicinal measures which have just been mentioned. In that year, however, Mikulicz sewed up a perforation in the lesser curva-

ture of the stomach, and although the result was unsuccessful, the operation at once became recognised as feasible. During the next ten years four other unsuccessful cases were recorded by Czerny (two), Wahl, and Mouisset; and it was not until 1892 that Kriege reported one which terminated in recovery. Since that time the operation has been so frequently practised that we have been able to collect eighty-seven cases, chiefly from English and American sources. Although the subject belongs to the domain of practical surgery, we think it advisable to give a short sketch of the operation and its results, for the benefit of those who may be called upon to perform it suddenly, without having had an opportunity of obtaining any special knowledge of the subject.

Indications for Operation.—The first point to be determined is whether the perforation is likely to be followed by local or general peritonitis. The latter condition is much the more common and is indicated by severe and prolonged collapse, general tenderness of the abdomen, retention of urine or difficulty of micturition, and disappearance of the liver dulness. In most cases the history shows that the stomach contained food at the time of the accident. Under these circumstances it may be accepted that the only hope of saving the patient's life is by the immediate performance of laparotomy. On the other hand, if the collapse is comparatively slight and the pain confined to the epigastrium, while the liver dulness persists and it can be absolutely determined that the stomach was empty at the time of perforation, the operation can be safely delayed for a time in the hope that spontaneous recovery may take place.

The question sometimes arises as to the advisability of operating when the peritonitis is already extensive and the patient nearly moribund. Each case must of course be judged upon its own merits; but it is wise to remember that while non-interference means certain death, there is always a prospect of recovery after operation. In several of the recorded cases the pulse, which was hardly perceptible before the operation, improved as soon as the peritoneum had been cleansed, and either the patient recovered or life was prolonged for several days.

Site of the Perforation. - Up to the present time almost

[!] Since this chapter was written a large number of additional cases have been published.

all those which have been operated upon have been examples of the acute perforating ulcer occurring in women between seventeen and twenty-seven years of age, and the puncture has usually been found at the cardiac end of the stomach near the lesser curvature, and, as a rule, upon the anterior surface. In a few instances, however, where the accident occurred in men about middle age, the perforation was discovered at the pyloric end near the lesser curvature. The difference in the location of acute and chronic ulcer has already been discussed (page 5). Perforation of the duodenum is most common in men about the age of thirty-five, and usually occurs upon the anterior or upper surface of the bowel within an inch and a half of the pylorus. It follows, therefore, that in young women there is a strong probability of the lesion being found in the cardiac extremity of the stomach, while in men the pyloric region and the duodenum are the parts which require primary attention.

Sketch of the Operation.—The operation may be divided into three stages: (1) the exposure of the stomach and discovery of the perforation; (2) the closure of the puncture; (3) the cleansing and drainage of the peritoneal cavity.

(1) The stomach is most easily exposed by a vertical incision carried downwards from just below the ribs for three or four inches, and slightly to the left of the median line of the abdomen. This incision can be subsequently enlarged, if necessary, either by extending it downwards, or by a transverse cut so as to divide the left rectus muscle. Having thus exposed the anterior surface of the stomach, a careful search must be made for the perforation. This is often indicated either by the presence of recent fibrinous adhesions, or by the formation of bubbles when the viscus is gently squeezed. Occasionally a sense of thickening in the wall of the stomach serves as a guide to the site of the opening. Should the organ be adherent to the under surface of the liver or other neighbouring part by recent lymph, these soft adhesions must be gently separated and the search continued. In some of the earlier operations the surgeon failed to discover the perforation, because he was afraid of disturbing the adhesions, a caution which defeated the main object of the operation and almost invariably proved fatal to the patient. If the anterior surface and upper border of the viscus are free from puncture, it must be everted and the posterior wall carefully searched in a similar fashion.

(2) As soon as the perforation has been found it should be brought as close to the wound as possible, while an attempt is made to empty the stomach of its contents. This may be performed either by means of a soft tube, inserted through the mouth, or more easily by gently expressing the chyme through the perforation itself, and immediately removing it with a swab. It is to be noticed that this manipulation of the stomach sometimes causes a disturbance of the pulse and respiration.

As soon as the organ has been emptied, an effort must be made to close the puncture. The ease with which this can be effected varies with its position and the existence of adhesions. If the ulcer is situated on the anterior wall near the middle, the perforation can easily be drawn to the surface and dealt with; but if it be near the cardia, considerable difficulty may be experienced. The size of the aperture in the stomach varies from one-sixth to one-half of an inch in diameter, and may either be clean cut or present ragged necrotic edges, in which case they may be pared with sharp scissors. Whenever it is possible, the best plan is to sew up the perforation with the Lembert suture, the ulcer being tucked in and the peritoneum closed over it by rows of stitches.

Excision of the ulcer has been practised on five occasions. the first successful case being recorded by Jowers. This method is undoubtedly the ideal one, since it not only closes the aperture, but, by removing the ulcer, prevents further mischief. These advantages are, however, more than counterbalanced by the tedious and difficult character of the operation. It would also appear that most acute ulcers heal readily after being closed in the manner described, as symptoms of recurrence after operation have been noted in only two instances. Where it is found impossible to close the perforation, several expedients may be adopted. In the first place the opening in the stomach may be stitched to the abdominal wound, and a drainage tube inserted into the cavity of the viscus. This method, which entails the formation of a temporary fistula, proved partially successful in the cases recorded by Haward, Parsons, and Maurice. Secondly, the hole may be either tamponed with gauze or closed by a piece of omentum which is stitched over The former has been performed with success by Hartmann, and the latter by Braun. Lastly, if no other procedure is possible, a tube must be inserted through the abdominal wound

to the vicinity of the perforation, to secure as free drainage as possible. Recovery after this latter operation has been reported

by Silcock.

(3) The third, and by no means the least important, part of the operation consists in cleansing the peritoneum from the products of inflammation, and in securing efficient drainage. The former may be achieved either by copious flushing of the cavity with sterile water, or by carefully wiping it out with an aseptic swab. As a rule, wiping is to be preferred to flushing, but in order to be successful it must be carried out in a systematic and efficient manner. The spot where the inflammatory products are chiefly apt to accumulate is the space which is situated between the left lobe of the liver, the diaphragm, stomach, and spleen. It is here that subphrenic abscess so often forms after the operation; and it is to prevent this complication that the whole cavity requires to be cleansed with such extreme care. Next in importance is the space between the right lobe of the liver and the diaphragm, where septic products also accumulate, owing to the suction action of respiration. Thirdly, the under surface of the liver and the sides of the gall-bladder require to be cleaned, and finally the lumbar regions, the cavity of the pelvis, and the interstices between the coils of gut. When the peritoneal cavity has thus been thoroughly wiped out, several gauze drains should be inserted with the view of protecting the dangerous localities aforementioned. Thus, one drain is passed down between the left lobe of the liver and the diaphragm, another between the right lobe and the diaphragm, a third to the under surface of the organ by the side of the gall-bladder. and a fourth into the pelvis. These drains can be removed between the fifth and seventh days if the case progresses well.

The after-treatment must be conducted upon the ordinary lines. It is usually wise to give the stomach complete rest for two days and to feed entirely by the rectum. To allay thirst, teaspoonful doses of water may be allowed, or an enema of hot water may be given. If no bad symptoms develop, a small quantity of albumen water may be administered by the mouth on the third day, after which the diet should be cautiously increased. The bowels must only be opened by enemata.

Sequelæ.—The immediate danger to life after the operation arises from *shock* or *peritonitis*, one or other of which was accountable for 72 per cent. of the deaths in the cases we have

collected. When life is prolonged for more than three days the chief danger to be apprehended is the formation of a localised abscess beneath the diaphragm, usually on the left side. This complication is due to inefficient cleansing and drainage of the subphrenic space, and may prove fatal at any period within the first two months, either by septicæmia or by secondary inflammation of the thoracic organs. It appears to have been responsible for about 14 per cent. of the deaths which took place after the first week, and was also noted in two cases which ultimately recovered. The greater attention which is now paid to the proper treatment of the peritoneum will probably banish subphrenic abscess from the catalogue of dangerous sequelæ. Pulmonary complications, such as pleurisy, empyema, pneumonia, and abscess of the lung, are almost invariably dependent upon the preceding affection, and may terminate fatally at any time between the tenth day and the second month (Horsley, Taylor). Intestinal obstruction is a rare result of laparotomy for perforation, and has only been observed in two cases. In one recorded by Kriege a piece of small bowel became strangulated by the omentum on the tenth day, while in the other, reported by Dean, death ensued two months after the suture of a duodenal ulcer from obstruction by a peritoneal band. Severe hamatemesis has only been observed after the operation in two cases, but in one of these it appears to have accelerated the fatal termination. Perforation of a second ulcer has been recorded by Stelzener and Mikulicz. When it is remembered how frequently the stomach is affected with multiple ulcers (page 3), the occasional occurrence of this phenomenon is hardly to be wondered at. It serves, however, to emphasise the necessity for manipulating the stomach with the greatest care and gentleness. In this connection it is interesting to observe that in two of the recorded cases the patient was suddenly attacked some days after the operation with abdominal pain and collapse of such severity as to suggest the possibility of secondary perforation. In each instance the abdominal wound was reopened and the stomach carefully examined, but no leakage could be discovered. It is possible that the symptoms may have arisen from gaseous distension of the viscus with stretching of some recent adhesions; but in all cases where such symptoms show themselves, and do not abate in a few hours, it is advisable to reopen the wound rather than run any risk from a second perforation or leakage.

Results.—The operation depends for its success or failure upon three factors: (1) the time which is permitted to elapse between the occurrence of the perforation and the performance of laparotomy; (2) the treatment of the hole in the stomach; (3) the care with which the peritoneum is cleansed.

(1) It has already been stated that the earlier the operation is performed the greater will be the chance of its success. This fact is well borne out by the results of an analysis of tifty-seven cases which were subjected to the same method of treatment.

Table 15.—Showing the Results of 57 Cases where the Perforation was closed by Lembert's Sutures

Interval between perforat and operation	ion	No. of cases	Recoveries	Deaths	Percentage mortality
1 to 6 hours .		9	, 5	41	33
6 ,, 12 .	• 1	15	11	4)	,,,,
12 ,, 18 ,, .		9	6	3+	50
18 ,, 24 ,, .	. 1	3	0	3)	00
More than 24 hours		21	3	18	86
Total	ij	57	25	32	

It will be seen that the mortality was only 33 per cent. for cases operated upon within the first twelve hours; but that it rose to 50 per cent. after another twelve hours; while after twenty-four hours had elapsed it was further augmented to 86 per cent.

(2) If the operation is to be successful, it is absolutely necessary that the perforation should be closed. This fact is well illustrated in the following table, which also indicates that the closure of the aperture by the Lembert suture is the one which gives the best results.

TABLE 16.—Showing an Analysis of 87 Cases treated in Various Ways.

Operation	No. of cases	Recoveries	Deaths	Percentage mortality
1. Open drainage	. 22	4	18	81
2. Perforation stitched to abdomina wound		0	5	100 (one case died after six weeks)
3. Ulcer excised .	. + 5	2	3	60
4. Ulcer sutured	. 55	24	81	56
Total	. 87	30	57	

(3) In most of the earlier operations the surgeon was content to flush out the peritoneal cavity without paying special attention to the subphrenic spaces and other regions of the abdomen, the insufficient cleansing of which constitutes the principal source of danger at a subsequent period. The importance of the knowledge which has been acquired by more extended experience in this and other points immediately connected with the operation is well illustrated by comparing the results of the earlier and later cases. Thus if the first fifty cases of laparotomy with suture of the ulcer be divided into two series of equal numbers, the results are found to be as follows:—

Table 17.—Showing a Comparison of the Results of the Earlier and Later Operations for Suture of a Perforated Gastric Ulcer

Series	Date of operation	Recoveries	Deaths	Percentage mortality
1st 25 cases . 2nd 25 cases .	1885 to 1894	5	20	80
	1894 ,, 1896	17	7	29

It will be observed that the mortality of the second series is only about one-third of the first. This result appears to justify the hope that in course of time the early performance of laparotomy, combined with skilful closure of the perforation and thorough cleansing of the peritoneum, will render the surgical treatment of perforated gastric ulcer one of the most successful operations of its kind.

Laparotomy for perforation of the duodenum has been performed seven times, with two recoveries (Dean, Dunn). The operation is essentially the same as that for gastric ulcer.

PART III

CHRONIC ULCER OF THE STOMACH AND DUODENUM

CHAPTER I

SYMPTOMATOLOGY

The onset of the disease is subject to considerable variation. In young women it sometimes commences quite suddenly by an attack of hæmatemesis, after recovery from which pain during the period of digestion, vomiting, or other symptoms manifest themselves. More commonly, however, discomfort after meals, with flatulence and constipation, is the main subject of complaint for several months, and it is not until the pain has become severe or hæmatemesis occurs that any suspicion is aroused as to the true nature of the case.

After the age of thirty a gastric ulcer usually commences in an insidious, manner with the symptoms of disordered digestion, which are gradually replaced by paroxysmal pain, and other phenomena that characterise the fully developed disease. Occasionally, after a period of general ill-health, a patient is suddenly attacked by pain and vomiting, which appear to be due to exposure to cold or to some indiscretion in diet; but instead of subsiding under treatment as in ordinary cases of gastric catarrh, the symptoms persist and are subsequently followed by hæmatemesis and other indications of ulceration.

In the majority of cases the symptoms of the complaint gradually increase until a climax is reached, after which they subside, often in inverse order of their occurrence, until complete recovery ensues. In other instances they continue with varying intensity for the greater part of life, sometimes dis-

appearing completely for weeks or months, at other times undergoing sudden exacerbations of such severity as to threaten immediate death. In every case the protracted nature of the complaint causes a serious impairment of the general nutrition, which may either lead to fatal exhaustion or favour the inception of phthisis or pneumonia. Finally, in a large proportion of the cases, life is brought to a sudden termination by the rupture of a blood-vessel, by perforation of the peritoneum, or by some other accident which is liable to attend progressive ulceration of the stomach.

Although the disease is apt to be accompanied by several manifestations of disordered digestion, there are three symptoms which from their prominence and frequency of occurrence must be considered as especially characteristic of its presence. These are severe pain, vomiting, and hæmatemesis.

Pain.—This constitutes the first in order of occurrence and also the most frequent and characteristic symptom of chronic gastric ulcer, and was present in every one of our 265 clinical cases of the disease.

It is variously described as a burning, boring, gnawing, or tearing sensation, which often gives rise to a sense of sickening depression or emptiness at the epigastrium; but whatever be the term by which different patients choose to express their sufferings they all agree that the pain is very severe, localised in the upper part of the abdomen, and usually paroxysmal in character. In nervous or debilitated subjects the paroxysm may be attended by syncope or convulsions.

Time of access.—During the early stages of the disease, and often throughout its entire course, the pain commences from five to twenty minutes after the ingestion of food, and increases until it reaches its maximum at the end of about two hours, after which it gradually declines and finally disappears at the conclusion of the period of gastric digestion. If vomiting happens to occur, the symptom subsides shortly after the stomach has been emptied of its contents. Sometimes the pain is experienced immediately the food has been swallowed, and in such cases the ulcer is often situated close to the cardiac orifice.

At intervals the pain becomes continuous, and may persist without intermission for many days or for even a week or two, after which it is either again replaced by the paroxysmal

variety or terminates in an attack of hæmorrhage or fatal perforation. This phenomenon is usually due to a rapid extension of the ulceration, accompanied by local peritonitis around the base of the sore.

In very chronic cases, and especially in those where the ulcer is situated near the pylorus and is adherent to the pancreas, the pain is chiefly experienced in the intervals of digestion, and is temporarily relieved by food and draughts of water. It is usually most severe at night between the hours of 11 p.m. and 5 a.m., and by preventing sleep tends to increase the exhaustion and mental depression from which the patient is already suffering. Lastly, in rare instances the pain exhibits a peculiar periodicity of recurrence. Thus cases are sometimes encountered in which it comes on regularly each day at the same hour, independently of food or of any other obvious cause.

Situation.—As a rule the pain is strictly localised to the

epigastrium, and is referred to a spot in the median line of the abdomen about one inch below the tip of the ensiform cartilage. affected area is circular in form, and varies from a quarter of an inch to two inches or even more in diameter. its actual extent being roughly proportionate to the size of the ulcer. Occasionally it is referred slightly to the left or to the right of the middle line. while in women who wear tight corsets the painful spot may be situated in the middle of the epigastrium, or even just above umbilieus. In exceptional

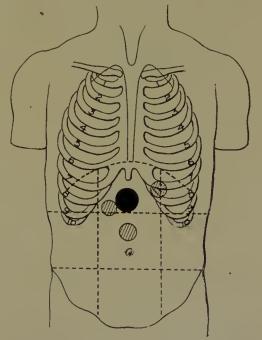


Fig. 44. The black disc represents the usual site of the epigastric pain and tenderness; the shaded spots their less frequent situation.

cases the chief seat of the pain is behind the lower end of the sternum or beneath the left costal arch.

Cruveilhier was the first to point out that many persons

suffering from chronic ulcer of the stomach complain of pain at one spot in the back after taking food, and subsequent investigations have shown that this symptom is of great diagnostic value. According to our experience this 'dorsal pain' develops several months after the epigastric variety, and when once it is established it remains fairly constant during the whole course of the disease. It is chiefly encountered where the ulcer occupies the posterior surface of the stomach, and has become adherent to the pancreas, diaphragm, or spine. The pain itself is usually described as a 'gnawing' or

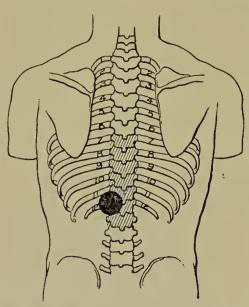


Fig. 45.—The black disc represents the usual site of the dorsal pain, but it may also occur at any spot in the shaded area.

'boring' sensation, which comes on coincidently with epigastrium that the and continues throughout the period of gastric digestion. It may be situated at any spot in the spine between the eighth dorsal and second lumbar vertebra, the most frequent position being between the eleventh dorsal and first lumbar vertebra and slightly to the left of the median line. Although the epigastric and dorsal pains usually coexist, they sometimes alternate with one another.

Brinton endeavoured to prove that the site of the

pain varied with the position of the disease in the stomach, the right hypochondrium being chiefly affected in cases of ulcer of the pylorus, and the left hypochondrium and left side of the spine when it occupied the cardiac end of the organ. Subsequent experience, however, has shown that the situation of the pain bears no constant relation to that of the ulcer.

Radiations.—In addition to these two fixed points, it is by no means unusual at the climax of an attack for the pain to become diffused over a much larger area or to be reflected along the course of certain nerves. Thus in severe paroxysms it often extends over the whole of the upper

part of the abdomen, round the sides of the chest or up the sternum towards the shoulders. In rare cases it is reflected along the nerves of the brachial, cervical, or lumbar plexuses. Occasionally the pain is produced by the direct invasion of a nerve by the ulcer. Thus Eisenlohr has recorded an instance in which violent pains of a neuralgic character were experienced over the lower portion of the left chest, owing to the implication of the thorax by an ulcer on the anterior wall of the stomach, and a somewhat similar one has been related by Pick. The adhesion of an ulcer to the diaphragm is sometimes followed by a sense of constriction round the base of the thorax, which is increased by deep inspiration and coughing, and by tenderness along the course of the phrenic nerves in the neck. Pain in the right shoulder during digestion or after physical exercise is often associated with the adhesion of an ulcer to the under surface of the liver, and Traube ascribes the attacks of dyspnea and precordial distress which sometimes follow the ingestion of food to irritation of the terminal branches of the pneumogastric nerves.

Effect of Food.—In all cases where the pain is paroxysmal in character the introduction of solid food into the stomach invariably excites an attack, and even when the symptom is more or less continuous it is always intensified after meals. The rapidity of onset, severity, and duration of the pain vary with the nature of the food; coarse and comparatively indigestible articles of diet like meat, pastry, and vegetables soon exciting violent suffering, while pulpy substances and liquid forms of nourishment may only be attended by a sense of discomfort. Hot foods are especially prone to produce it, and in some cases ice or draughts of cold water exert an equally deleterious effect. It is to be observed that tea, beer, and spirits are almost always injurious during the early stages of the disease; but in very chronic cases the pain may be temporarily relieved by the ingestion of warm tea and weak brandy (Travers). Milk, whether given pure or diluted with water, hardly ever gives rise to pain, and this fact is of considerable value in the diagnosis of ulceration from functional disorders of the stomach.

Effect of Pressure.—Another characteristic feature of the epigastric pain is that it is always increased by pressure, and many patients complain that they are unable to wear corsets

or other tight articles of clothing on account of 'a sore spot in the stomach.' To ascertain the existence of localised tenderness, the knees should be bent over a pillow to relax the abdominal muscles, and the patient's attention distracted, while graduated pressure is made with the tip of the finger over different parts of the epigastrium. The examination is best made when the stomach is empty, and must be conducted with the greatest care and gentleness, since careless or longcontinued manipulation not only occasions much suffering, but is apt to excite hæmatemesis or even to produce perforation of the organ. As a rule pain ensues immediately the pressure is applied, but sometimes many seconds may elapse before it When the dorsal pain is present it can usually be excited by manipulation of the epigastrium, the sensation appearing to the patient to pass from the sore spot in the abdomen straight through to the back; but direct pressure applied to the spine itself neither induces nor increases the symptom.

Cases have been recorded in which pressure upon the epigastrium has relieved rather than aggravated the pain (Cruveilhier, Henoch, Brinton, Miquel). In these instances it is probable that the ulcer was not situated directly under the finger, so that the effect of the pressure was to tilt up the base of the sore and thus to remove it from the opposed surface of the stomach. A similar effect can sometimes be produced by the application of lateral pressure to the sides of the chest over the lower ribs.

The passage of a weak electric current through the stomach soon after a meal affords a still more delicate test for the presence of an open ulcer. The experiment is performed by placing the pad connected with the positive pole of a galvanic battery over the lower dorsal spine, and gradually increasing the strength of the current until it can be felt as a slight tingling sensation when the negative electrode is applied to the left hypochondrium. When the latter is placed immediately over the ulcer severe pain is excited, and if the sensitive area is mapped out upon the skin, it will be found to correspond in position with the tender spot to which the paroxysmal pain is referred.

Effect of Posture.—The fact that the intensity of the pain varies with the position of the body was first pointed out by

SYMPTOMATOLOGY

189

Osborne and afterwards much insisted upon by Brinton. This latter writer endeavoured to prove that the site of the ulcer could be accurately determined by observing the posture which the patient assumed during an access of pain. Thus, if he preferred to lie upon his back, the disease occupied the anterior wall of the stomach; or if the prone posture gave most relief, the posterior wall was affected by the ulcer; while if he reclined upon his right or left side, the lesion involved the cardia or pylorus respectively. That these different forms of decubitus are occasionally observed is certainly correct; but on the other hand so many exceptions to Brinton's law are encountered that at the present day very little attention is bestowed upon the subject. As a matter of fact most persons suffering from an ulcer of the stomach prefer to lie upon their back during the painful paroxysm, while cases have been recorded of ulcer of the anterior wall where the prone posture was always adopted (Chambers, Nasse). It is supposed that the preference accorded to the dorsal decubitus is due to the relaxation of the abdominal muscles which is best obtained in this posture; but it is quite as possible that rest upon the back diminishes the movements of the organ and also relieves the strain upon any adhesions that are present. This appears to be confirmed by the fact that in all cases physical exertion greatly increases the pain, while in many cases, where the ulcer is adherent to the liver or pancreas, violent movements of the body will give rise to a severe paroxysm even when the organ is free from food.

Mental emotions and intellectual overstrain are extremely apt to excite an attack which differs in no particular from that which follows the ingestion of solid food; while any sudden impairment of the general health, such as may be produced by an attack of influenza, tonsillitis, or of some specific fever, is almost always accompanied by a notable exacerbation of the gastric phenomena.

In most women the pain of a gastric ulcer is greatly augmented before each menstrual period, and at the same time the epigastric tenderness becomes increased and the vomiting is more urgent. Hæmatemesis is also apt to occur at these times. As a rule, the exacerbation is observed from twenty-four to forty-eight hours before the appearance of the discharge, and subsides as soon as it has become well

established. These symptoms are quite distinct from those that accompany painful menstruation.

Variations in the intensity of the pain are frequently observed during the course of the disease, and may be due to several causes. A rapid subsidence of the symptom is the usual result of the substitution of milk for the ordinary mixed diet; but it may also ensue from sloughing of an exposed nerve (Habershon) or from the relief of tension which is afforded by an attack of hæmorrhage. A gradual but steady improvement usually accompanies the process of cicatrisation.

In this connection, however, it must be observed that an ulcer of the stomach may lose its sensibility in the same manner that a chronic sore upon the leg often becomes quite painless; so it must not be concluded that because the pain has disappeared the disease has necessarily been cured. The fallacy of such a belief was recently brought home to us by the case of a middle-aged man who had suffered for many years with the symptoms of chronic ulcer of the stomach. Under medical treatment the pain gradually subsided, the tenderness almost disappeared, and his general health improved to such an extent that we acquiesced in his oftrepeated assertion that his disease was cured. later, however, he died from an attack of pneumonia, and when the stomach was examined a large chronic ulcer, which showed no signs of healing, was found on the posterior wall of the organ. Cases of this kind are not very uncommon, and perhaps account for many of the so-called 'relapsing' ulcers, where the symptoms subside for a time and subsequently recur with all their former severity.

Severe exacerbations of pain are commonly due to a rapid extension of the ulceration, with involvement of the peritoneum, and for this reason they are especially apt to herald the approach of hæmorrhage or perforation. A recrudescence after a long period of immunity is often a sign that the cicatrix has been attacked by secondary ulceration.

Cause of the Pain.—The paroxysmal variety, which occurs during the period of digestion, probably owes its origin to the co-operation of several factors. In the first place the regularity with which the symptom follows the ingestion of solid food, and the rapid manner in which it subsides when the patient is placed upon a liquid diet, show that direct irritation of the ulcer plays

an important part in its production. In the second place, the movements of the organ which occur after meals probably increase the severity of the suffering by inducing traction upon the edges and base of the sore. Thirdly, the tendency of the pain gradually to increase until it reaches a climax, notwithstanding the reduction of the food to a pulpy consistence, can only be explained by the presence of an abnormally acid gastric juice, which irritates the sore and may produce spasmodic contraction of the pyloric portion of the organ; while in those cases where pain is chiefly experienced between the meals, it owes its origin almost entirely to the hypersecretion of acid. Finally, the access which is often observed immediately prior to perforation is probably caused by inflammation of the serous membrane at the base of the ulcer.

Vomiting.—This symptom is less constant than the preceding, but it was observed at some period or other during the course of the disease in nearly 72 per cent. of our clinical cases.

In its most typical form it ensues as soon as the pain has become severe, and has the effect of curtailing the crisis by ridding the stomach of its irritant contents. When the disease has existed for a considerable time, it often occurs at intervals and lasts for several days, during which time the patient is unable to retain any kind of nourishment. In other instances the emesis chiefly takes place between the meals, while in those cases where the ulceration affects the orifices of the stomach it resembles in its general features that which accompanies stricture of the œsophagus or stenosis of the pylorus. It will therefore be seen that the symptom may be indicative of several different conditions, each of which merits a separate description.

(1) When vomiting is due merely to the presence of an ulcer in the stomach, it occurs from fifteen minutes to two hours after a meal, and usually at the climax of the paroxysm which the food has provoked. The emesis is preceded by acidity, flatulence, or nausea, and is more an act of regurgitation than of actual vomiting, the rejection of the gastric contents being seldom accompanied by straining or nausea. As a rule, it secures the complete evacuation of the stomach, with the result that the pain rapidly subsides. The

ejecta consist of the food taken at the last meal, which has undergone partial digestion. After filtration the fluid is found to have a total acidity varying from 55 to 90, and to contain an excess of hydrochloric acid along with peptones, albumoses, and other products of digestion. Occasionally traces of blood may be detected by appropriate tests, while in very rare instances the erosion of some neighbouring organ is demonstrated by the discovery of minute pieces of liver (Miquel), pancreas, or spleen (Sangalli) in the vomit. Rest in bed and the substitution of milk for solid food arc almost invariably followed by the subsidence of the symptom.

- (2) In long-standing cases, and especially in those where the ulcer affects the pyloric region and is adherent to the pancreas or liver, vomiting often occurs during the night or in the intervals of digestion. This form of emesis is usually preceded by pain in the abdomen and back, flatulence, acidity, nausea, and other symptoms of hypersecretion. The ejecta vary in amount from two to twelve ounces or more, and consist of an opaline acid fluid which is often tinged yellow or green from admixture with bile. Chemical examination shows that it consists essentially of gastric juice containing a slight excess of hydrochloric acid and a small amount of mucus and undigested starch. When this variety has once become established, it usually continues until the end.
- (3) Secondary catarrh of the gastric mucous membrane is a frequent result of chronic ulceration, and is responsible for another form of vomiting. In most of these cases the patient experiences a sense of weight and oppression at the chest one or two hours after a meal, with distension of the abdomen, flatulent eructations, loss of appetite and nausea, followed by severe retching. The ejecta are small in quantity, viscid, and often bile-stained, and consist of mucus secreted by the inflamed stomach, mixed with ropy saliva. From time to time subacute attacks of gastric catarrh are apt to supervene, and give rise to incessant retching and vomiting of alkaline mucus. Occasionally diacetic acid is formed in the stomach, the absorption of which, it is believed, may produce drowsiness or coma.
- (4) An ulcer situated at the pylorus is very prone to cause obstruction of the orifice during the process of healing. When the degree of stenosis is only slight, vomiting usually occurs

about two hours after a meal; but as soon as the stomach has become dilated, retention of the food takes place, and the emesis is repeated at irregular intervals. Under these conditions the vomit is excessive in quantity, sour-smelling, and contains an excess of hydrochloric acid. Ulceration of the cardiac orifice is accompanied by pain and difficulty of deglutition, and by regurgitation of the food in an unchanged state.

Vomiting is always a serious symptom in chronic ulcer of the stomach, and is often directly responsible for the fatal issue. In all cases the rejection of food, even if it occurs only once a day, gradually leads to loss of flesh, anemia, and general debility. a state of health which favours the inception of phthisis and other infective diseases. Catarrhal conditions of the stomach are particularly dangerous; for not only does the incessant retching prevent the administration of nourishment, but the severe physical exhaustion which it entails may induce fatal syncope or coma. Lastly, a severe attack of vomiting may produce such violent movements of the stomach as to tear away the adhesions formed between the base of the ulcer and some neighbouring organ, and thus give rise to fatal perforation. Cases of this description have been recorded by Fox, Sabrazès, and others, and we have known the same accident to follow the administration of an emetic.

Hæmorrhage (Hæmatemesis and Melæna).—It is probable that every case of chronic gastric ulcer is accompanied by some degree of hæmorrhage; but if only slight oozing occurs, the blood is entirely eliminated by the bowel in a condition so changed as to escape recognition on a casual examination of the fæces. The fact that an ulcer often bleeds in this way is well illustrated by two cases recorded by Einhorn, in which the contents of the stomach, extracted after a meal, were found quite unexpectedly to contain a considerable quantity of blood. It is obvious, therefore, that the detection of hæmorrhage depends to a great extent upon the care with which the vomit and evacuations are examined, and the recognition of this fact helps to explain the divergent views which have been expressed by different writers as to the frequency of hæmatemesis in chronic ulceration of the stomach. Thus Lebert noted its occurrence in 80 per cent. of his clinical cases; Ewald in about 50 percent.; Gerhardt in 47 per cent.; Müller in 30 per cent.; Brinton and Witte in 29 per cent.; and Miquel in 17 per cent. In our own series of

265 cases, either hæmatemesis or melæna was observed in 188, or in about 71 per cent.

The process of ulceration being as a rule very slow, several months or even years may elapse before the disease penetrates sufficiently deep to destroy one of the larger gastric vessels; and where the subjective phenomena consist principally of flatulence and discomfort after meals, an attack of hæmatemesis may prove the first symptom to attract the serious attention of the patient or his medical attendant. In most instances the hæmorrhage presents itself without any premonition; but occasionally it is preceded for a short time by continuous pain in the epigastrium or back, or by paroxysms of unusual severity. The sudden recurrence of pain after a long interval is especially apt to herald an attack, since in such cases there is either an acute extension of the disease or a rapid erosion of a cicatrix (Cruveilhier).

As a rule the bleeding occurs shortly after a meal, the increased determination of blood to the stomach which accompanies the process of digestion, combined with the stretching of the wall of the viscus, being the immediate cause of the rupture of the diseased vessel. Occasionally an excess of alcohol, severe vomiting, straining at stool, or even pressure upon the epigastrium appears to be the determinate factor in the production of the hæmorrhage. Cornillon observed hæmatemesis after the performance of lavage, and we have known severe bleeding follow exploration of the stomach with a soft tube.

The liability to hæmorrhage is increased by any condition which raises the pressure of the blood in the vessels of the stomach or materially alters the composition of the fluid. Thus repeated attacks of hæmatemesis are extremely common when the ulcer is complicated with disease of the heart, interstitial nephritis, cirrhosis of the liver, enlargement of the spleen, purpura, scurvy, or by pneumonia or other febrile complaints. A sudden increase in the determination of blood to the organ, such as occurs immediately prior to each menstrual epoch, is also apt to give rise to severe bleeding, and in not a few women who suffer from chronic ulcer of the stomach hæmatemesis only makes its appearance at these times. The following case is a good illustration of this interesting fact.

Case XXVII. A lady who had always enjoyed excellent health began at the age of 38 to suffer from pain after food and other

symptoms of chronic ulcer of the stomach, which always became much worse a few days before the period. At the end of eighteen months, on the day before the period was due, she suddenly grew faint and vomited about sixteen ounces of bright blood, after which the menses were suppressed for three months. For the next eleven months the symptoms were kept in check by careful dieting, but she continued liable to attacks of pain whenever she over-exerted herself or had any special anxiety. At the end of this time, without any obvious cause, she was again attacked by severe hæmorrhage just before the menstrual period. During the course of the next three years the bleeding recurred on four occasions, each time within fortyeight hours of the appearance of the discharge, which continued regular but much diminished in quantity. After the sixth attack she gradually lost the epigastric pain and tenderness, gained considerably in weight, and for the last four years has been able to take all forms of food without any discomfort, so that there is every reason to believe that the ulcer has undergone cicatrisation.

In the majority of cases the hæmorrhage manifests itself by vomiting of blood, followed by melæna. Occasionally, however, hæmatemesis is absent, and the whole of the effused blood escapes by the bowel, while in rare instances the bleeding is so profuse as to cause fatal syncope without any external evidence of the accident.

The clinical aspect of gastric hemorrhage varies according to the quantity of blood which is lost, and may be appropriately described under the terms 'slight,' 'moderate,' and 'severe.'

(1) Slight hæmorrhage.—In this category are included those small but frequent losses of blood which ensue from the destruction of the minute vessels of the mucous and submucous coats of the stomach. The actual amount probably seldem exceeds two or three ounces. If vomiting occurs at once, the food appears to be streaked with blood or mixed with small clots; but when it is postponed for a short time, the blood undergoes digestion, with the result that it acquires a brown colour and a gritty appearance (coffee-grounds). In this process its albuminous constituents are converted into a form of globulin, while the hæmoglobin is changed into hæmatin. Several substances besides blood impart a brownish tint to the vomit, as, for example, red wines, coffee, and the various preparations of iron, while a similar coloration of the stools may be produced by the administration of calomel and bismuth.

In every ease, therefore, the vomit and sides of the vessel

should be carefully examined for the presence of brownish-black masses or flakes, or, failing this, the ejecta should be filtered and search be made among the residue upon the paper. Under the microscope these small particles are seen to be composed of granules of pigment mixed with shrunken red corpuscles; and if there is any doubt as to the nature of the colouring matter, the question may be set at rest by the following process devised by Korczinski and Jaworski. A small quantity of the material is mixed in a porcelain dish with a few grains of chlorate of potassium and a drop of hydrochloric acid, and

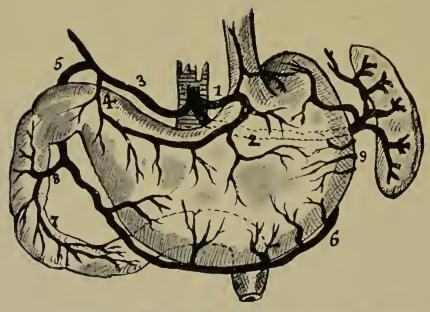


Fig. 46.—Diagram of the stomach and duodenum, showing their arterial blood supply. 1, coronary artery; 2, splenic; 3, hepatic; 4, pyloric; 5, gastro-duodenal; 6, right gastro-epiploic; 7, 8, superior pancreatico-duodenal; 9, left gastro-epiploic.

gently evaporated to dryness. If any altered blood is present, the addition of a dilute solution of ferrocyanide of potassium to the residue gives rise to an intense blue colour. The filtrate can also be tested for blood by means of fresh tincture of guaiacum and ozonic ether, and if it contains an excess of hydrochloric acid it may deposit crystals of hæmin after standing for a few days.

This variety of hæmorrhage is seldom accompanied by any noticeable symptoms, although occasionally the patient may complain of nausca and giddiness, or become suddenly pale

and faint. The frequent recurrence of slight hæmorrhages may produce a condition of intense anæmia, the cause of which easily escapes notice unless the stools are regularly and systematically examined.

(2) Moderate hamorrhage.—This is usually due to the erosion of a medium-sized artery or vein in the submucous or subperitoneal tissue of the stomach, but it also occasionally ensues from the destruction of some neighbouring organ, such as the pancreas, liver, or spleen. The quantity of blood which is vomited varies from six to eighteen ounces or more; but this seldom represents the whole amount, since a notable proportion always finds its way into the intestine. Its colour

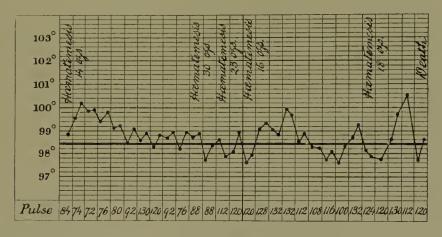


Fig. 47.—Temperature chart in a case of chronic gastric ulcer with repeated hæmatemesis. The patient vomited 101 ounces of blood in a fortnight.

differs according to the rapidity of the effusion and the length of time it has remained in the stomach, rapid hæmorrhage and immediate vomiting being evidenced by the rejection of bright clotted blood, while a more tardy expulsion renders it darker in colour and more fluid in consistence. In most instances the hæmatemesis occurs without any premonition, but occasionally it is preceded by a feeling of heat or fulness at the epigastrium, palpitation of the heart, or a peculiar taste in the mouth, followed by nausea, faintness, or even convulsions.

The symptoms which ensue from the loss of blood are similar to those already described in cases of hamorrhage from acute ulcer (p. 135). The patient becomes markedly anomic,

restless, and thirsty, and subsequently cold and prostrated. The pulse is somewhat quick and jerky in character, the temperature depressed, and giddiness, faintness, noises in the ears, and dimness of vision are frequent sources of complaint. The urine is pale and abundant, and may contain a trace of albumen. After the shock has passed off, there is often a slight elevation of temperature for several weeks.

A copious hæmorrhage often relieves the epigastric pain and tenderness for a time, and occasionally the symptoms of ulceration gradually disappear as the patient recovers from his loss of blood. More commonly, however, the relief is only transient, and as soon as he commences to take solid food the former symptoms are renewed.

In nearly 40 per cent. of our cases the hæmorrhage recurred within a week of its apparent cessation, and in 73 per cent. within twelve months. At first sight these figures may seem to exaggerate the danger of a relapse; but it must be remembered that they are entirely derived from hospital practice, where patients habitually ignore all instructions concerning diet as soon as they are well enough to resume their former mode of life.

When the hæmorrhage recurs within a short time, it is due either to insufficient clotting of the blood or to dislodgment of the thrombus which had temporarily closed the mouth of the vessel. In the former case the injured artery continues to leak in an intermittent manner, so that the patient vomits altered blood at intervals, but in diminishing quantities, until eventually the ejecta merely exhibit a brownish tint. On the other hand, the displacement or disintegration of the thrombus is followed at once by vomiting of a large amount of bright blood. In recurrent hæmatemesis the quantity of blood which is lost is sometimes extraordinary. Thus in the case from which fig. 47 was taken, the patient vomited 101 fluid ounces in a fortnight, and probably lost at least half as much again by the bowel.

When the bleeding returns after a lapse of several days it is almost invariably due to dislodgment of the thrombus. In some cases this arises from a natural recovery of arterial tension, but more often it is the result of the injudicious administration of stimulants, of excitement, or of physical exercise. We have known several instances where the slight

exertion entailed by a medical examination was sufficient to bring on another attack, and one in which fatal bleeding was excited by the patient getting out of bed in order to micturate.

When the hæmorrhage recurs after a long interval it is usually found that another vessel has become involved by the disease, and, since the deeper the ulcer the larger the arteries with which it comes into contact, the second attack is usually more serious than the first. In rare cases severe hæmatemesis occurs long after the symptoms of the complaint have subsided and the ulcer has apparently healed. This may be due either to the rupture of a vessel in the centre of the cicatrix (case xxv.), to the development of an acute ulcer, or to the fact that the disease had become 'latent' and had not been cured.

In acute ulceration of the stomach, the loss of blood, however great it may have been, is usually repaired rapidly and completely, but in the chronic form of the disease the consequences are far more serious. Each attack leaves the patient more feeble and anæmic, with a diminished appetite and lessened powers of digestion. In some instances improvement gradually takes place, but more often, and especially in persons above middle age, the nutrition becomes progressively impaired, and the case proceeds to a fatal termination.

(3) Excessive hæmorrhage (l'hémorrhagie foudroyante).— Hæmorrhage of such severity as to prove immediately fatal is comparatively rare. Brinton believed that it took place in about 5 per cent. of all cases of gastric ulcer; but in our own series it was only observed in 3.4 per cent. The vessels which are usually affected are the coronary or splenic, but in rare instances the blood is furnished by the spleen, abdominal aorta, or even by the heart itself (Finny). Occasionally the cause of the bleeding is to be found in the rupture of a small aneurism at the base of the ulcer (Powell, West, Rasmussen, Welch). all cases the patient suddenly becomes blanched, falls to the ground, and rapidly loses consciousness. Sometimes convulsions occur. As a rule hæmatemesis is observed before death, but the shock may be so intense as to paralyse the nervous centres and so inhibit vomiting. In such the stomach and intestines are found filled with blood at the autopsy, or a large fluctuating tumour in the epigastrium, due to distension

of the stomach with clot, may be detected before life becomes extinct.

Perforation.—The most serious symptom of chronic ulceration of the stomach is perforation of the general cavity of the peritoneum. The frequency of this accident has been the subject of some discussion. Brinton found sixty-seven examples of perforation in 257 cases of open ulcer which he collected from various sources; and, acting upon the assumption that 50 per cent. of all ulcers heal spontaneously, he concluded that one case in every seven or eight, or about 13.4 per cent., terminated in this manner. This estimate has been generally accepted by subsequent writers, notwithstanding the fact that the calculation is vitiated by several obvious sources of error. Thus, in the first place, it is probable that the proportion of recoveries from the disease is far more than 50 per cent., a modest estimate being that two out of every three ulcers heal during life (p. 34). Secondly, the method of collecting isolated cases of the disease is open to serious objection, since it is only when some striking event, like perforation or fatal hæmorrhage, occurs that the case is deemed worthy of publication. Lastly, many of the authors whose statistics Brinton accepted made no distinction between perforation with general peritonitis and that which gave rise to localised abscess.

Welch came to the conclusion, from the study of a large number of autopsies, that the accident occurs in about $6\frac{1}{2}$ per cent. of all cases, while Engel estimates its frequency at $5\frac{1}{2}$ per

cent., and Miquel at about 2 per cent.

Owing to our inadequate knowledge of the frequency with which cicatrisation takes place, all computations based upon post-mortem statistics must necessarily be imperfect. A more trustworthy method is to observe the number of times perforation occurs in a large series of clinical cases. Thus, out of the 252 cases which came under the care of Lebert, nine, or 3.5 per cent., died from perforative peritonitis, and in our own series of 265 cases of chronic ulcer the death rate from this cause amounted to $5\frac{1}{2}$ per cent. If due allowance be made for those that are lost sight of before a cure has been effected, and for that not inconsiderable class where secondary ulceration occurs in the cicatrix, it is probable that about 7 per cent. of all cases terminate in perforation.

The accident is most frequent in females, in whom it also

occurs at an earlier period of life than in males. Thus Berthold found perforation in 3·1 per cent. of his cases of gastric ulcer in males, and in 9·7 per cent. of the cases in females. According to our statistics the average age in men is forty-three years, and in women twenty-seven. This difference obviously depends upon the mode of origin of the ulcer in the two sexes, since in women the complaint often commences in an acute manner at a comparatively early age (fifteen to twenty-five years), while in men it develops insidiously after the age of thirty.

Perforation of the stomach may either occur quite suddenly without any premonitory sign, or it may be preceded for several days by an increase in the pain and vomiting, or by an attack of hæmatemesis. As a rule it takes place from the gradual destruction of the tissues by the process of ulceration, but in some cases it is due to the rupture of the base of the disease by a violent muscular effort. Thus it has been known to result from severe retching or vomiting, and Faber has recorded an instance where it followed the emesis produced by voluntary irritation of the pharynx. Bouillaud observed it from straining at stool; Ewald, during the performance of lavage; while we have known it occur both from the passage of a bougie, and after the administration of an emetic. other instances, again, it is probable that adhesions between the base of the ulcer and the liver were ruptured by severe jolting of the body, by gymnastic exercises, by pressure upon the epigastrium (Henoch), or by massage of the abdomen (Jackson).

The general symptoms which follow perforation of the stomach have already been described (p. 142), so that it only remains to notice a few peculiarities of the accident in chronic ulcer. In old people, and in those who have been greatly debilitated by the gastric complaint, death may occur from general peritonitis without any of the ordinary symptoms presenting themselves. In such the abdominal pain is very slight, or may be absent altogether; there is no elevation of the temperature, and the only phenomena that attract attention are a quick feeble pulse, a dry tongue, and excessive prostration; even gaseous distension of the intestines is often wanting, and actual retraction of the abdomen has been observed (Bouveret). Suppression of urine occasionally occurs (Sedgwick), and paralysis of the lower extremities has been recorded by Talamon.

Localised abscess from perforation will be described among the sequelæ of gastric ulcer (Part IV. Chap. v.).

Disorders of Digestion.—In addition to the characteristic pain and vomiting after food, many cases also suffer from symptoms which arise from functional disorder of the digestive organs. The most prominent of these are discomfort after

meals, flatulence, acidity, nausea, and constipation.

Discomfort after food is most frequently due to chronic gastric catarrh. It usually comes on from one to two hours after a meal, and gives rise to a sense of oppression at the chest accompanied by difficulty of respiration and flatulent distension of the abdomen. In some cases these symptoms are associated with an asthmatic condition which is not relieved until a quantity of gas has been expelled from the stomach or bowel. Violent palpitation of the heart, throbbing in the head, giddiness, and nausea are also occasionally observed.

Acidity may arise either from an excessive secretion of the gastric juice or from fermentation of the food. The former condition is by far the more common, owing to the frequent association of hypersecretion with ulcer of the stomach. The symptom usually makes itself felt about an hour and a half after a meal, and occasions a cramping pain behind the lower end of the sternum, followed by the regurgitation of an acid fluid into the pharynx and mouth. These phenomena also occur in the intervals of digestion, and more especially during the night, when they are apt to excite retching and vomiting. This form of acidity is temporarily relieved by the ingestion of milk or by draughts of alkaline fluids.

Fermentation of the food chiefly takes place when the ulcer has narrowed the pylorus and given rise to secondary catarrh of the stomach. Under these conditions acidity and flatulence are experienced one or two hours after food, and are

followed by nausea and vomiting.

Constipation is an almost invariable symptom of chronic ulcer, and probably owes its existence to several causes. In the first place, the severe pain which follows the ingestion of solid food necessitates the employment of a milk diet, which in itself favours the condition, while the occurrence of vomiting still further deprives the bowel of the normal stimulus arising from the presence of chyme. Secondly, the severe irritation of the stomach caused by the ulcer induces a reflex paresis

of the coats of the intestine, so that its peristaltic movements are greatly diminished or entirely suppressed. In the third place, alterations in the shape of the stomach, adhesions of its walls to neighbouring viscera, and chronic peritonitis about the base of the disease, all tend to trammel the movements of the organ and to prevent the due propulsion of the food along the alimentary tract. Finally, it is possible that the excessive acidity of the gastric contents neutralises the intestinal fluids and prevents the bile from exerting its laxative action.

In very chronic ulcers associated with hypersecretion, attacks of diarrhea are apt to alternate with periods of constipation, owing to the supervention of secondary catarrh of the colon, while in those rare cases where the ulcer forms a fistulous communication with the large bowel, undigested food is often evacuated immediately after meals (lientery).

The appearance of the tongue varies with the state of the gastric mucous membrane. In uncomplicated cases it is red, clean, and moist; but when gastritis is present it becomes thickly coated with a greyish-white fur, and the breath acquires a sour or fœtid odour. Occasionally, during an exacerbation of the complaint, small aphthous ulcers make their appearance upon the dorsum and along the margins of the organ (Abercrombie, Henoch).

The appetite is usually unimpaired during the early stages of the disease, although the patient is afraid to indulge his inclination on account of the distress which follows a meal; but when the pain becomes continuous or the vomiting excessive, the desire tor food is temporarily lost. When hyperacidity complicates the disease, the appetite is sometimes ravenous, the excessive acidity of the secretion appearing to stimulate the terminal filaments of the gastric nerves. With the advent of chronic hypersecretion, on the other hand, the appetite often becomes capricious, or there may be curious cravings for certain articles, such as raw ham, sardines, or tomatoes. Chronic catarrh of the stomach is almost always accompanied by a diminished desire for food; and in advanced cases complete anorexia, like that which attends carcinoma of the stomach, may be present.

Thirst is seldom a noticeable symptom during the early stages of the complaint, but it usually follows the establishment

of hypersecretion, and is then chiefly experienced after meals or during the night. It is relieved by alkaline drinks and increased by acids.

An excessive secretion of saliva is sometimes observed in long-standing cases, and occasionally gives rise to much annoyance. Contrary to the statement of Bamberger, there is often at first an increase in the amount of sulphocyanide of potassium, and we have even found it abundant after severe hæmorrhage, and when the patient has been confined to a liquid diet for some time. This fact is of some importance in the diagnosis between simple ulcer and cancer, for in the latter complaint the salt tends to disappear from the saliva after the course of a few months. When emaciation and debility supervene, the salt gradually diminishes in quantity, and its final disappearance usually indicates an early termination of the case.

For a variable period after the disease has declared itself, the general health continues good, and there is no abatement of the physical or mental powers. With the progress of time, however, the pain and vomiting produce a steady loss of flesh and impairment of strength. The rapidity and extent of the emaciation are proportionate to the severity of the symptoms; and it may usually be observed that whenever a remission takes place the patient begins to regain weight. With the advent of hypersecretion or chronic gastric catarrh, loss of flesh becomes a marked feature, and may eventually rival in degree the emaciation of cancer or tuberculosis. When the complaint attacks young children, the growth of the body is seriously interfered with, and Chvostek has recorded a case in which a patient at the age of eighteen resembled in size and stature a boy of ten.

As long as the general symptoms of the complaint remain of moderate severity and no hæmorrhage takes place, the mucous membranes retain their natural colour, and to all outward appearance the patient may seem to be perfectly well; but as soon as the nutrition becomes seriously impaired or losses of blood occur, the face becomes pale and the lips and conjunctive markedly anamic. In most instances these signs never develop to any great extent, but vary from time to time according to the progress of the disease; but if hæmorrhage is of frequent occurrence, the skin gradually acquires a wax-like pallor, dyspnœa is experienced on exertion, and the legs

and feet become ædematons. Should hæmatemesis be absent and all the blood be eliminated by the bowel, the cause of the anæmia is very liable to be overlooked; and numerous cases have come under our notice in which neglect to examine the stools was responsible for an erroneous diagnosis of pernicious anæmia. Occasionally this last-named disease actually does occur during the course of ulceration of the stomach (Litten); while in very chronic cases we have known a true cachexia develop, attended by the same degree of prostration and loss of flesh as occurs in cancer of the organ.

Examination of the blood reveals a diminution in the number of the red corpuscles and in the amount of hæmoglobin, but the number of the white cells may be slightly increased, especially during the period of digestion.

Amenorrhæa is much less common in chronic than in acute ulcer, and is chiefly encountered in women below the age of thirty years. When the ulcer occurs at a later period of life, the catamenia continue regular though often scanty. A severe attack of hæmorrhage from the stomach may be followed by suppression of the menses for a month or two, or even by their permanent disappearance.

The constant suffering engendered by the complaint usually produces great irritability and depression of spirits, which sometimes develop into melancholia, while persons who are predisposed to nervous affections often exhibit symptoms of hypochondriasis or hysteria, which may quite eclipse those of the original disease and thus lead to an erroneous diagnosis. When chronic ulcer occurs in women of a highly nervous temperament, a condition of neurasthenia occasionally supervenes, and may continue long after the gastric phenomena have subsided. In such cases the patient complains of intense physical and mental exhaustion, which is increased by exertion, and is often so severe as to confine her to bed. The bowels are obstinately confined, and a sense of sickening depression at the epigastrium, with faintness or nansea, often follows an evacuation. Owing to want of exercise, the muscles of the extremities waste, and in bad cases contractures or bed sores may develop.

In uncomplicated cases the temperature of the body is never elevated, and with the progress of exhaustion it usually sinks below the normal. Temporary elevations occur after attacks of hæmorrhage, and if the loss of blood has been severe the temperature may vary between 99° and 101° F. for several weeks. With this exception a febrile state is usually indicative of some secondary affection such as tuberculosis, perigastritis, or abscess.

The condition of the urine varies according to the functional activity of the stomach. In uncomplicated cases it is quite normal, but if hypersecretion is present it may exhibit several important changes. Owing to the deficient absorption consequent upon the vomiting, the quantity of the secretion is much diminished, and only fifteen to twenty ounces may be voided in the twenty-four hours. The colour is pale, and sometimes the fluid has a slightly milky appearance when freshly passed, while its density is increased owing to concentration and to an excess of its saline constituents. The acidity is invariably diminished, especially after meals, and in advanced cases the fluid is often permanently alkaline. Phosphates, urates, and sometimes crystals of uric acid are deposited on stand-Sometimes peptones can be detected in it, and transient albuminuria is not infrequent. The daily excretion of urea is increased, but the percentage of chlorides is notably diminished (Gluzinski, Rosenthal, Sticker). After perforation of the stomach retention of urine is a common symptom, and suppression an occasional one (Sedgwick); albuminuria may occur after severe hæmorrhage (Quincke).

The occasional occurrence of acetonuria and diaceturia has already been noticed. These substances are the result of abnormal fermentations in the stomach, associated with catarrh of the mucous membrane. They impart a sweet odour to the breath and the urine, and their absorption into the general circulation is supposed to give rise to abdominal pain, vomiting, dryness of the tongue, thirst, great prostration, drowsiness, and coma. The two latter symptoms may possibly arise from the presence of acetone in the blood, but the others are indistinguishable from those which commonly accompany an attack of gastric catarrh.

Physical Signs.—The most important sign of an ulcer of the stomach is the presence of a tender area in the epigastric region just below the tip of the ensiform cartilage. In itself this phenomenon has little diagnostic value, as it is also met with in inflammatory diseases of the stomach and colon, con-

gestion and cirrhosis of the liver, pericarditis, and diaphragmatic pleurisy. When, however, these various affections can be eliminated, while the tender area remains localised in the same position, and corresponds with the spot to which the paroxysmal pain after food is referred, it affords valuable confirmatory evidence of the existence of an ulcer.

Hyperesthesia of the skin over the epigastrium and upper part of the abdomen has recently been brought forward by certain theorists as an important indication of the disease. As a matter of fact this condition is infinitely more common in functional disorders of the stomach and colon, and in carcinoma and tuberculosis of the peritoneum, than in gastric ulcer, so that as a sign of the latter complaint it is not only worthless but actually misleading.

Occasionally the superficial reflexes over the epigastrium and left hypochondrium are remarkably increased.

Excessive pulsation of the abdominal aorta is sometimes observed, especially during a paroxysm of pain. It is most pronounced after the disease has existed for some time, and appears to be due to a dilatation of the aorta from reflex paralysis of its vaso-motor nerves, and is analogous to the dilatation of the temporal and carotid arteries observed in certain forms of migraine (Rosenbach).

According to Peter the temperature of the skin over the epigastrium is usually elevated in chronic ulcer of the stomach, but our own observations have not tended to support this statement.

It is sometimes possible to confirm the diagnosis in a doubtful case by administering substances which give rise to pain by irritation of the surface of the ulcer. For this purpose two or three teaspoonfuls of salt dissolved in a tumblerful of water is the most effective, although chlorate of potassium and dilute solutions of hydrochloric acid (3 to 4 per cent.) may also be employed. The draught is given in the early morning when the stomach is empty, and if an open ulcer is present it is usually followed by localised pain in the epigastrium and sometimes by vomiting.

The passage of a weak electric current through the stomach has already been mentioned as a means of detecting the position of an ulcer, and determining whether it has undergone cicatrisation (p. 190).

The determination of the size of the stomach has a certain amount of value in the localisation of the disease. Thus if the organ is greatly dilated it may be presumed that the ulcer has given rise to stenosis of the pylorus, while in cases where it is markedly contracted and is accompanied by dysphagia and other signs of esophageal obstruction the disease is probably situated close to the cardiac orifice.

It is usually taught that one of the principal distinctions between simple ulcer and cancer is the absence in the former complaint of a palpable tumour inconnection with the stomach. Although the statement is substantially correct, it sometimes happens that a too strict observance of this rule is responsible for a serious error in diagnosis.

There are six conditions which, in order of frequency, may give rise to a tumour connected with a simple ulcer, namely, (1) hypertrophy of the tissues about the pylorus, (2) adhesion of a neighbouring organ to the base of the disease, (3) displacement of the pancreas, (4) an encysted collection of pus, (5) a thick cicatrix, (6) enlarged glands.¹

(1) Excessive thickening of the tissues around an ulcer of the pylorus has been observed in several cases. Thus Clarke records the case of a man, forty-five years of age, who had suffered for some time from pain after food, and vomiting. The right side of the epigastrium appeared somewhat fuller than the left, and contained a tumour the size of an orange, which was extremely tender. The stomach was moderately dilated. After death, which occurred from perforative peritonitis, a tumour about the size of two fists was found at the pyloric end of the organ, to which the neighbouring viscera were united by adhesions. The mass was composed of pale tough fibrous tissue, which surrounded two deep ulcers with ragged walls, one of which had perforated the peritoneum. Microscopical examination showed that the tumour was composed entirely of inflammatory tissue. The pyloric orifice was stenosed and the stomach much dilated. A very similar condition has come under our own observation.

Case XXVIII. A middle-aged woman was admitted into the London Hospital for cancer of the stomach. She was greatly emaciated and cachectic, and suffered from constant retching and vomit-

Out of sixteen cases of tumour with ulcer collected by Reinhard, six were due to thickening of the pylorus, and six to adhesions with other organs.

ing. The illness was supposed to have lasted for about a year, but there had never been any hæmatemesis or melæna. On examination the stomach was found to be greatly dilated, and its peristaltic movements were visible through the thin abdominal walls. Immediately above and to the right of the navel there was a prominent tumour the size of a large egg, which moved on respiration, was dull on percussion, and very tender. These signs, taken in conjunction with the general appearance of the patient, appeared to indicate the existence of carcinoma of the pylorus; but an examination of the vomit showed that it contained a large amount of free hydrochloric acid. The case was consequently diagnosed as one of chronic ulcer with inflammatory thickening. The patient died after she had been three days in the hospital, and at the post-mortem examination a deep ulcer was found on the posterior wall of the stomach near the pylorus, surrounded by great induration of the tissues, which the microscope proved was purely inflammatory in origin.

Rosenbach has called attention to a species of phantom tumour which arises from spasmodic contraction of the wall of the stomach in the neighbourhood of the ulcer; and in one instance of this nature, recorded by an American writer, the muscular coat in the pyloric end of the viscus was so greatly hypertrophied that it formed a sausage-shaped tumour, which could be felt to harden and relax when grasped by the hand. Finally, it must be mentioned that occasionally the tumour may be composed of the entire stomach, as in cases of plastic perigastritis or where the organ is firmly contracted from stenosis of the cardiac orifice.

(2) The structures which most often form a tumour by adhesion to the ulcer are the liver, gall-bladder, omentum, and skin. In the case of the liver the tumour may consist either of a hydatid cyst (Netter) or a gumma, or, as in a case which came under our own observation, of the left lobe of the organ, which was twisted and distorted by cirrhosis. Adhesion of an ulcer to the gall-bladder is chiefly encountered when the disease is situated in the first part of the duodenum. In some of these cases the tumour appears tense and elastic owing to distension of the bladder with bile and mucus, while in others it is hard and nodular from the presence of numerous calcul. in its interior.

Adhesion of the omentum to the base of an ulcer occasionally takes place when the disease is situated upon the anterior wall of the stomach, and may give rise to a palpable

tumour. Beer has recorded an interesting case of this description, in which the existence of a mass in connection with the stomach led to an erroneous diagnosis of carcinoma, and at the present time we have a young lady under our care who has a well-marked tumour in the epigastrium as the result of perforation of the stomach some months ago.

In those rare cases where an ulcer becomes adherent to the abdominal parietes, a hard swelling may develop in the subcutaneous tissue near the umbilicus prior to the establishment

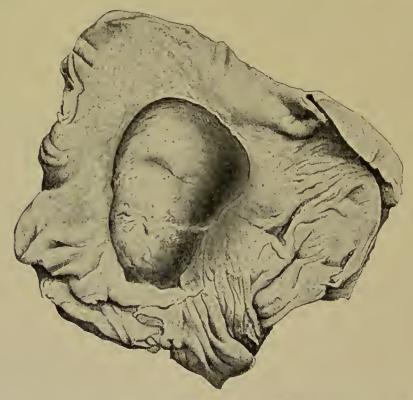


Fig. 48.—Drawing of the posterior wall of a stomach, showing a large chronic ulcer, through which the pancreas projects in the form of a tumour. (London Hospital Museum.)

of a gastric fistula. Pick has recorded an instance in which the disease penetrated the diaphragm and produced a large brawny tumour upon the left side of the chest, which was regarded as a sarcoma of the ribs until its real nature was ascertained at the autopsy.

(3) Tumours formed by the pancreas are much less frequent than the preceding, and the only example of this kind with which we are personally acquainted is shown in fig. 48, where it may be seen that a chronic ulcer has its base composed entirely of the pancreas, while through its centre a large piece of the gland projects into the cavity of the stomach.

Kollmar cites the case of a man who died with extreme cachexia, an abdominal tumour, and other signs of cancer of the stomach. At the autopsy the naked-eye appearances of the disease seemed to confirm the diagnosis, but when the tumour was incised it was found to consist of a pot-shaped ulcer with thickened edges, through the base of which the head of the pancreas projected.

If a tumour of this description can puzzle the pathologist, it is hardly surprising that the surgeon is occasionally confused by the condition which he detects upon a digital exploration of the stomach through an exploratory incision. The difficulties that attend this method of diagnosis are well illustrated by a case recorded by Palawski, where an exploratory operation was undertaken on account of continuous pain, vomiting, and hæmatemesis. When the abdomen was opened the stomach was found adherent to the neighbouring viscera, and the retroperitoneal glands were much enlarged. The finger was then inserted through an incision in the anterior wall of the stomach, and a large cauliflower growth was felt upon the posterior wall. Carcinoma was accordingly diagnosed, and the wound closed; but at the autopsy the supposititious growth was found to consist of the pancreas, which projected through the base of a simple chronic ulcer.

- (4) An encysted collection of pus over the anterior aspect of the stomach has frequently been mistaken for a malignant growth, and in one of our cases an abscess with thick walls was found in the omentum between the lower border of the stomach and the transverse colon.
- (5) A tumour due to a chronic ulcer or a thick cicatrix is very rarely observed, and only one case has come under our own notice. In this instance a middle-aged man was admitted into the hospital for what was supposed to be a cancerous stricture of the bowel. The only history that could be obtained was that he had suffered from obstinate constipation, with frequent vomiting, for several months. The stomach was greatly dilated, and in the pyloric region an oval tender tumour was detected. The vomit was free from hydrochloric acid. The patient died in a few hours, and at the autopsy

a thick annular scar was found to encircle the pyloric orifice, the lumen of which was greatly contracted.

(6) Enlargement of the pyloric glands not infrequently ensues from chronic ulceration of the stomach, and it would appear, from a case published by Dreschfeld, that in rare cases they may give rise to a tumour which can be detected during life.

Examination of the Contents of the Stomach.—Although a soft tube is frequently used by Continental physicians for the extraction of the gastric contents where ulcer is suspected, we consider it a most dangerous procedure, and never employ it except under special circumstances.

Examination of the vomit shows that in about two-thirds of all cases of chronic ulcer of the stomach the amount of hydrochloric acid is increased and varies from 0.25 to 0.5 per cent., while in the remaining third the presence of the free acid can usually be detected by appropriate tests. If emesis occurs when the stomach is empty (i.e. during the night), the ejecta may consist almost entirely of gastric juice, the acid constituent of which is either normal or slightly increased in amount (hypersecretion). When chronic catarrh complicates the disease, the vomit usually consists of neutral or alkaline mucus.

Duodenal Ulcer.—The clinical history in cases of duodenal ulcer is usually very unsatisfactory. Thus out of the 151 cases collected by Perry and Shaw no fewer than ninety-one, or 60 per cent., failed to exhibit any symptoms of importance prior to the onset of the fatal hæmorrhage or perforation, while about 53 per cent. of those analysed by Cullen presented a similar condition of latency. These writers, however, included both the acute and chronic forms of the disease in their statistics. Out of our own series of forty-three cases of chronic ulcer of the duodenum, thirty-two, or 74 per cent., had suffered from abdominal pain, vomiting, or hæmorrhage for some time before death.

Pain was recorded in thirty out of the forty-three cases, and was most severe when the ulcer was immediately contiguous to the pylorus and upon the posterior wall of the bowel. It varied in character from a sense of uneasiness to violent and prolonged suffering, and was chiefly referred to the right hypochondrium or to the region of the umbilicus. In the majority of cases its onset is quite independent of

the ingestion of food, and is often most severe at a time when the stomach is empty. Occasionally it is excited by exertion or by some expiratory effort, such as laughing, sneezing, or coughing. In its most typical form it ensues from two and a half to four hours after a meal containing solid food, and radiates from just below the right costal arch over the upper part of the abdomen and back. Localisation to one spot is seldom observed, nor is there usually any pain in the back analogous to the dorsal pain met with in gastric ulcer. Acidity, flatulence, and other symptoms of a functional disorder of digestion frequently precede and accompany the incidence of the pain.

Vomiting was present in only eight cases, or in about 20 per cent. When it occurs it bears no relation to the severity of the pain. If the ulcer has given rise to stenosis of the bowel, the character of the vomiting resembles that which results from dilatation of the stomach. As a rule, the bowels are constipated, but diarrhea was observed in about 8 per cent. of our cases.

The occurrence of hæmorrhage from the stomach or bowel is the most important indication of the existence of an ulcer. This symptom was recorded in seventeen out of the forty-three cases, or in about 40 per cent. In five out of the seventeen there was apparently hæmatemesis without melæna, in five it was associated with the passage of altered blood by the bowel, and in the remaining seven melæna was the only sign of the hæmorrhage. These facts appear to disprove the usual statement that melæna is far more common than hæmatemesis, but it must be remembered that while vomiting of blood always attracts the attention of a patient and his medical attendant, the presence of black blood in the stools is frequently overlooked; indeed, many persons are brought to the verge of death from internal hæmorrhage without being in the least conscious that they are passing blood in their stools.

The symptoms of hemorrhage are identical with those already described in the case of gastric ulcer, and, as in that disease, death may occur without either hematemesis or melana. It is interesting to remark that in almost every instance of fatal bleeding the ulcer is situated upon the posterior or inner wall of the bowel within four inches of the pylorus.

Perforation occurred in twenty-three, or 53·5 per cent., of our cases, and was followed by general peritonitis in twenty-one and by an abscess in two instances. In almost every case the disease was situated upon the anterior wall of the intestine. The accident may occur quite suddenly, or it may be preceded by severe pain, vomiting, and constipation. It has been known to follow violent exercise (Stillwell), straining at stool (Spitta), or a fall upon the abdomen (Hutchinson). The symptoms of the accident do not differ from those of perforation of the stomach, except perhaps that vomiting is somewhat more frequent. Death usually ensues from general peritonitis within thirty-six hours, though occasionally life is prolonged until the fourth day. Sudden death from perforation has been recorded by Murchison and Spitta.

CHAPTER II

$THE \ CLINICAL \ VARIETIES \ OF \ CHRONIC \ GASTRIC \ ULCER$

THE description of the disease given in the preceding chapter refers to the ordinary form of gastric ulcer, in which the occurrence of severe pain after meals, vomiting, or attacks of hæmorrhage renders the diagnosis a matter of ease But it has been known since the time of and certainty. Abercrombie and Cruveilhier that certain cases present themselves in which these characteristic symptoms are either absent altogether, or one or other of them is so pronounced as to obscure the real nature of the complaint; and it was the recognition of this important fact that induced Lebert to describe ten different varieties of the disease. examination, however, of this and other classifications that have been made, shows that in most instances they are based upon such minute differences in the various symptoms that they are more apt to confuse than to aid the practitioner in his diagnosis. We therefore consider that it will be sufficient to indicate five forms of chronic ulcer of the stomach in which, owing to the special predominance of some particular symptom, such as pain, vomiting, dyspepsia, hæmorrhage, or cachexia, the detection of the ulcer becomes a matter of more than ordinary difficulty.

(1) The gastralgic form.—This constitutes the commonest variety of the disease, and is characterised by intense pain, which may exist for a length of time without vomiting or hæmatemesis.

The subjects of this form of the malady are usually men from thirty to forty-five years of age, of a tall spare build, very nervous or excitable in temperament, and who are often liable to attacks of neuralgia in different parts of the body. In not a few instances of this kind there is a strong family predisposition to phthisis, and we have known several cases which eventually fell victims to a rapid form of pulmonary tuberculosis. Persons who have lived in malarious districts, whether they have actually suffered from the fever or not, seem to be particularly liable to excessive pain if attacked by ulcer of the stomach.

The pain is severe from the outset, and gradually increases in intensity as the disease progresses. At first it may occur only after food, but it soon develops a tendency to appear at all times of the day or during the night. Sometimes an attack will persist for several days or even weeks, being temporarily relieved by the ingestion of food, while at others it either disappears completely or is replaced by a sense of soreness and discomfort. Although the epigastric region is the chief seat of the suffering, the pain often radiates all over the abdomen, as well as upwards towards the clavicles and round the chest to the interscapular region. During an attack the patient will often roll upon the floor in agony, or recline over the back of a chair as though he were suffering from angina pectoris. Among the minor symptoms of the complaint are acidity, flatulence, deficient appetite, and obstinate constipation. Loss of flesh is almost always present, and great depression of spirits, and even melancholia with a suicidal tendency, are apt to ensue. These various symptoms either gradually subside and finally disappear altogether, or are followed after the lapse of some months by hæmatemesis.

Case XXIX. An officer in the Army, 39 years of age, consulted us on account of severe abdominal pain from which he had suffered for nearly two years. His history showed that he had always enjoyed good health until he was attacked, at the age of 25, with a mild form of fever in India. He had never had syphilis, and there was no family predisposition to any special disease. At its onset the pain only occurred after luncheon, but it subsequently ensued after every meal, and within four or five months became more or less continuous. There was considerable acidity and flatulence, the bowels were constipated, and he had lost over two stones in weight during the last eighteen months. There had never been any vomiting or hamatemesis.

On examination the patient was found to be very thin and somewhat anæmic. The thoracic organs were normal, the liver and spleen not enlarged, and there was neither sugar nor albumen in the urine. The abdomen was firmly retracted, and the whole of the

epigastric region was very tender on pressure; but no tumour could be detected. The pain was chiefly referred to a spot just above the umbilicus, from which it radiated all over the abdomen and back. When distended with soda-water the lower border of the stomach was found to reach just below the navel. The gall-bladder could not be felt, nor was there any pain upon pressure over its usual site.

After remaining in this condition for about nine months, he was attacked by profuse hæmatemesis, which recurred on two subsequent occasions. Eventually, about five years from the commencement of the complaint, the pain gradually diminished, and at the present time, with the exception of occasional symptoms of dyspepsia and consti-

pation, he enjoys good health.

Case XXX. A man, aged 32 years, consulted us for what he called 'neuralgia of the stomach.' It appeared that, five years before, he had had an attack of acute gout in the left great toe, but with this exception his health had been extremely good. The gastric symptoms had commenced two years before his visit to us, and at first consisted of flatulence and discomfort immediately after meals, but in the course of a few months they were replaced by violent attacks of pain in the intervals of digestion. Recently the pain had recurred regularly each afternoon, whether he indulged in lunch or not, and after lasting for an hour or two terminated in the expulsion of a large quantity of gas from the stomach or bowel. Sometimes acidity was a prominent feature of the attack. From time to time these symptoms would disappear for a month or two, and would then recur with equal suddenness and with all their former severity. He had not suffered from syphilis or malaria, vomiting had never occurred, and his nights were not disturbed by pain or acidity.

On examination the stomach was found to be slightly dilated and the epigastric region very tender on pressure. The gall-bladder could not be felt, and there was no history of biliary colic or jaundice. The other organs of the body were quite normal. Under medical treatment and strict dieting the attacks became much less severe; but at the end of five months the pain suddenly recurred, and remained constant for several weeks, and was described as being at times 'excruciating.' This state of affairs continued for about eighteen months, attacks of severe suffering alternating with intervals of complete relief, when during one of these remissions he suddenly vomited a large quantity of blood. The bleeding occurred on two subsequent occasions, but eventually the pain disappeared and the patient made a complete recovery.

Several explanations may be offered for the occurrence of such severe pain in ulcer of the stomach. In the majority of the cases it is probable that the symptom arises from irritation of the sore by the hyperacid gastric juice which usually accompanies this form of the disease; for it may be observed that the pain is apt to ensue during the intervals of digestion and is temporarily relieved by draughts of milk or alkaline waters. In other instances it seems to be due to the involvement of an important nerve by the ulcer or to the coexistence of hyperæsthesia of the gastric mucous membrane; while not infrequently local inflammation of the serous coat of the viscus, the formation of an abscess, or ulceration of a neighbouring organ is found after death to have been the cause of the excessive suffering.

Finally, it must be mentioned that neurotic persons are apt unintentionally to exaggerate their sensations, while those who have suffered from malaria and syphilis are extremely liable to develop neuralgia from any source of irritation.

(2) The catarrhal or 'vomiting' form.—In this variety the chief symptoms of the complaint are due to the presence of secondary catarrh of the stomach. It is principally encountered in men of middle age, and is characterised by periodic attacks of discomfort after meals, followed by prolonged and severe vomiting, which persists for some time and then gradually disappears. In elderly people the exhaustion entailed by the disease frequently gives rise to a fatal issue.

Case XXXI. A gentleman, at the age of 30, began to suffer from attacks of severe vomiting in the autumn of each year. The complaint always commenced quite suddenly about the end of October, with distension and flatulence after food, followed in a few hours by violent retching and vomiting, which continued for several days and prevented the administration of any nourishment by the mouth. The bowels were much confined, the tongue foul, and great prostration accompanied each attack. During the rest of the year, with the exception of a little discomfort after solid food, he usually felt quite well, and was able to pursue his occupation, which involved a good deal of travelling and mental strain. When we saw him he was suffering from his sixth attack, and for nearly a fortnight had vomited every kind of nourishment, whether liquid or solid, as soon as he swallowed it. There was no complaint of pain, but the epigastrium was slightly distended and tender on pressure. The bowels were very confined, the tongue foul, and the temperature subnormal. The vomit, in the intervals of taking food, consisted of alkaline and bile-stained mucus. A week later he was seized with violent hæmatemesis, which soon proved fatal.

The entire absence of pain in the above case, combined with the regular recurrence of the seizures, led us to diagnose subacute gastric catarrh. It was therefore with no slight mortification that we learned the fatal termination of the illness, which in all probability was due to the erosion of a large vessel by a chronic ulcer of the stomach. Of course it may be objected, as there was no autopsy, that the hæmorrhage may have been due to some other morbid condition; but the absence of any history of intemperance, combined with the normal condition of the liver and other important viscera, renders this supposition very unlikely. In the following cases the presence of ulceration was placed beyond all doubt.

Case XXXII. A gentleman, upwards of 80 years of age, was attacked with severe vomiting which lasted for several weeks and led to great exhaustion. The emesis was chiefly excited by the administration of nourishment, but occasionally he would be seized with violent retching, which only subsided upon the rejection of some alkaline mucus. There was no complaint of abdominal pain, and there were no signs of disease in any important organ of the body. After recovery from this illness the bowels were very confined, and the appetite was somewhat diminished. About a year afterwards he began to experience discomfort after meals, with gaseous distension of the abdomen, and nausea, but there was no pain. A few days later vomiting set in, and he became unable to retain any nourishment. When we saw him, at the end of about ten days, he was extremely prostrate, and expressed the greatest repugnance to food. The tongue was thickly coated with a creamy fur, the pulse was quick and feeble, and the temperature of the body depressed. The stomach was dilated, the great curvature reaching more than two inches below the level of the navel. There was no tenderness on pressure, and no tumour could be detected. The bowels responded easily to aperient medicines, and there was no stricture of the rectum or any evidence of obstruction of the intestine. The vomiting continued unchecked by treatment, and the patient rapidly sank from exhaustion.

On post-mortem examination the stomach was found to be moderately dilated, and its mucous surface showed signs of chronic inflammation. Close to the pylorus, on the posterior surface of the viscus, there was a scar of considerable size, which had given rise to slight contraction of the orifice, while a little below, and to its inner side, there was a chronic ulcer the size of a two-shilling piece. About the centre of the stomach, near the lesser curvature, were three other ulcers of recent formation, which had exposed the muscular coat.

Case XXXIII. A labourer, aged 46, was admitted into the London Temperanee Hospital 'for uncontrollable vomiting,' from which he had suffered for nearly a fortnight. He was so extremely prostrate that no detailed history could be obtained, but it appeared that he had been liable to similar seizures for about three years. The attacks would come on every few months, with discomfort and flatulence after meals, followed in about forty-eight hours by severe retching and vomiting, which prevented him from taking any kind of food. There was never any pain, but the bowels were very confined. He had always been a temperate man, and there was no history of either hæmatemesis or melæna. The ejecta eonsisted entirely of mucus mixed with yellow bile. The stomach was found to be slightly dilated, and pressure upon the epigastrium gave rise to nausea. There was no tumour. Resort was had to rectal feeding, but the patient died in about twenty-four hours from cardiac failure.

At the autopsy the stomach was found to be somewhat dilated. Immediately contiguous to the pylorus, on the posterior wall of the organ, was a chronic ulcer which had almost cicatrised. The inner surface of the viscus was covered with a tenacious layer of mucus, and when this was removed the mucous membrane was seen to be swollen and opaque, and spotted here and there with small interstitial hæmorrhages.

These three cases are sufficient to bear out the statement previously made, that pain may be completely absent in ulcer of the stomach, and that the only source of complaint may be severe and recurrent attacks of gastric catarrh.

(3) The dyspeptic or 'latent' form.—The principal symptoms of this variety are discomfort and flatulent distension of the stomach after meals, with occasional acidity, nausea, or attacks of vomiting, while in many instances the only indication of any gastric trouble is to be found in the statement of the patient that 'meat is apt to disagree.' Constant acidity, if combined with the discovery of an excess of hydrochloric acid after a test meal, might suggest the presence of an ulcer; but in the majority of the cases no organic mischief is ever suspected until either hæmorrhage occurs or the disease is discovered after death. Cases of this description are by no means uncommon, so that a single example will suffice.

Case XXXIV. A man, 54 years of age, was admitted into the London Hospital for chronic phthisis, from which he had suffered for nearly four years. He made no complaint of indigestion, but on being questioned admitted that he often had flatulence after meals,

and was unable to take beef, because it seemed to 'lie heavy on the chest.' There was no acidity, nausea, or vomiting, and examination of the abdomen only showed a slight degree of dilutation of the stomach. At the end of about six weeks the patient was attacked by hamoptysis, from which he died, and at the post-mortem examination, in addition to the tubercular mischief in the lung, a chronic ulcer three inches in diameter was found near the pylorus on the posterior wall of the stomach.

(4) The hamorrhagic form.—Certain cases of gastric ulcer are characterised by repeated attacks of hæmorrhage, to which the other symptoms are quite subordinate. In some instances profuse hæmatemesis occurs every two or three months for several years, and in the intervals the patient merely complains of discomfort or flatulence after meals, while in others small hæmorrhages take place every few days, or the stools are frequently found to contain altered blood. The constant loss of blood is apt to produce a very serious effect upon the general health. The skin and mucous membranes become intensely anæmic, and the patient grows so feeble that the slightest exertion brings on an attack of palpitation or syncope. The appetite fails, the legs and feet become ædematous, and loss of memory, failure of vision, insomnia, and other symptoms of cerebral anæmia present themselves. condition may persist, with occasional intermissions, for several months, and may terminate in recovery after all hope has been abandoned; or life may be brought to a sudden termination by cardiac failure or by a more profuse hæmorrhage than usual. In one of our cases, which ultimately recovered, eight severe attacks of hæmatemesis occurred within six months, while in another it was calculated that the patient must have lost more than three-quarters of the total quantity of blood she possessed at the commencement of her disease.

Excessive hæmorrhage is particularly common when an ulcer of the stomach is associated with valvular disease of the heart, interstitial nephritis, cirrhosis of the liver, or enlargement of the spleen, and sometimes appears to be induced by the scorbutic condition which results from the long-continued use of boiled or sterilised milk. In some women profuse hæmatemesis occurs at each catamenial period.

(5) The cachectic form.—There is an important variety of gastric ulcer in which the principal symptoms of the complaint

are emaciation, cachexia, and extreme debility. As a rule these phenomena only appear after the disease has existed for a considerable time, and has given rise either to stenosis of the pylorus, or to deformity of the body of the stomach by cicatrices, or to permanent hypersecretion. Occasionally, however, cachexia and loss of flesh precede the development of abdominal pain and vomiting, and under these circumstances the case is easily mistaken for one of carcinoma; in fact, it is only by the discovery of free hydrochloric acid in the contents of the stomach that a differential diagnosis can be made. These cases usually run a somewhat rapid course, and more often terminate from exhaustion than from perforation or hæmorrhage.

Case XXXV. A labouring man, about 42 years of age, was admitted into the hospital for 'pernicious anæmia.' He stated that his health had been failing for about twelve months, during which time he had lost nearly two stones in weight, and had become so weak that he was unable to work. For the last three months his friends had remarked the pallor of his face, and he had found that the slightest exertion gave rise to palpitation and shortness of breath. Latterly swelling of the feet had occurred. The appetite was much diminished, and after taking food he suffered from oppression at the chest, distension of the abdomen, and flatulence. There had never been any vomiting or hæmatemesis. The bowels were very confined.

On examination the patient was found to be extremely emaciated and markedly anæmie. The tongue was coated with a grey fur, the breath sour, and the temperature of the body subnormal. The stomach extended to the level of the navel, and pressure over the epigastrium gave rise to discomfort; but no localised tenderness or tumour could be detected. The other viscera appeared perfectly normal. The blood showed a notable decrease in the number of red corpuseles and of hæmoglobin, but the changes in the corpuscles characteristic of pernicious anæmia were not observed. Under treatment he improved somewhat, and the appetite returned, but there was little increase in weight or strength. After about two months he suddenly became much worse, and complained of pain at the epigastrium, and vomited his food. The temperature rose to 101° F., an indefinite tumour was felt in the epigastrium, and he succumbed to exhaustion at the end of a week.

At the autopsy a large chronic ulcer was found on the posterior wall of the stomach, about three inches from the pylorus and close to the lesser curvature. The greater part of its base was firmly

adherent to the pancreas, but at the upper margin of the gland it had perforated all the coats of the stomach and had given rise to a small encysted collection of pus.

The foregoing case was regarded during life as an example of cancer of the posterior wall of the stomach, but the autopsy showed that the disease was of a benign nature.

CHAPTER III

DURATION AND PROGNOSIS

THERE are several reasons why it is impossible to determine with any degree of accuracy the duration of a chronic ulcer of the stomach.

In the first place, it is always a difficult matter to ascertain from the patient's history the exact date at which the symptoms commenced, while it is equally impossible to fix the period at which cicatrisation becomes complete. The progress of the complaint also varies in a remarkable manner with the position of the ulcer, those which are situated on the anterior wall or at the upper margin of the organ being apt to terminate comparatively early by perforation or hæmorrhage, while such as occupy the posterior surface often contract adhesions with the pancreas and remain stationary for a number of years. Again, the course of the disease is notably influenced by diet and regimen, so that no statistical inquiry is of any value unless the cases which have remained continuously under treatment are carefully differentiated from those that have been allowed to pursue their course unchecked. Lastly, an ulcer may contract the pylorus or cardia, may be attacked by cancer, or give rise to hypersecretion or secondary catarrh of the stomach, any of which sequelæ tend to replace the original disease as the determinate factor in the ultimate progress of the case.

Lebert endeavoured to determine the mean duration of the complaint by analysing a series of cases according to the time at which death or probable cure occurred, and came to the conclusion that the majority of ulcers persist from three to five years. Bouveret, on the other hand, believes that the actual duration of the malady does not exceed two years, while in our own series of cases the average duration was about four years and eight months.

Table 18.—Showing the Duration of the Disease in 110 Cases of Chronic Gastric Ulcer (Lebert).

	Fatal	Non-fatal	Total	Percentage
Latent	7	9	16	14.55
1 to 6 months.	0	8	8	7.27
6 , 12 ,	š	7	12	10.9
1 9 *******	6	16	22	20.0
3 ,, 6 ,, · .	$1\overset{\circ}{4}$	15	29	26.36
6 , 10 ,	5	4	9	8.19
10 , 15 ,	4	2	6	5.45
15 ,, 20 ,,	2	3	5	4.55
20 ,, 35 ,,	1	2	3	2.73
Total	44	66	110	100

Cases have been recorded in which thirty to forty years elapsed between the initial hæmorrhage and death, during the whole of which time the patient was hardly ever free from pain or vomiting after meals.

Prognosis.—The mortality from chronic ulcer of the stomach has been variously estimated by different writers. Brinton concluded that only about one-half of the cases were amenable to cure, and this statement has been recently endorsed by Debove and Rémond. On the other hand, Lebert observed a fatal termination in only about 8 per cent. of his clinical cases, while Rosenheim fixes the mortality at 10 per cent.

The explanation of this divergence of opinion is by no means difficult. It has already been shown that the material upon which Brinton based his conclusions was eminently untrustworthy, owing to his method of collecting isolated cases; while his estimate of the number of healed ulcers found after death is much below the truth, the proportion of scars to open ulcers being at least three to one. Again, a careful examination of the statistics dealing with the mortality from gastric ulcer reveals the fact that many writers make no distinction between death with gastric ulcer and death from gastric ulcer in other words, they include in their figures a large number of cases in which the immediate cause of death was only remotely connected with the local lesion. Thus in 40 per cent. of the fatal cases tabulated by Debove and Rémond, the patients really succumbed to tuberculosis, while in other statistics deaths from perforation and hæmorrhage are not differentiated from those which resulted from valvular disease of the heart, pneumonia, or other intercurrent diseases.

That all persons who suffer from chronic ulceration of the stomach do not die from affections immediately connected with it is well shown in the following table, where it is seen that about 26 per cent. of our cases succumbed to some intercurrent disease.

Table 19.—Showing the Cause of Death in 100 Cases where an Open Ulcer was found at the Autopsy

Perforation with ger	neral pe	ritoni	tis				32,	
Perforation with loc	calised a	bsces	s.				7	
Hæmorrhage .							17	
Exhaustion .							15	74 per cent.
$\mathbf{Fistule} \left\{ egin{aligned} \mathbf{Gastro-tho} \\ \mathbf{Gastro-col} \end{aligned} ight.$	oracic ic	1)					2	
Cancer secondary to	o ulcer				,		1	
Cardiac disease .							5	
D 3	Tubero	ele .	11					
Pulmonary disease	Pneum Pleuris	onia sy	$\begin{bmatrix} 6 \\ 1 \end{bmatrix}$	•	٠	•	18	
Tubercular peritoni	tis .						1	
Apoplexy							1	
Bright's disease								
Total .							$\overline{100}$	

It must always be remembered that the progress of the disease depends to a great extent upon its stage of development at the time when the patient comes under medical treatment, and the care with which he carries out the prescribed diet. This fact is of the greatest importance, and helps to explain in a great measure the difference in the death rate recorded by various writers. Thus we find that among our hospital clinical cases 17 per cent. succumbed immediately to the complaint, while in our private practice, where the necessary treatment was carried out in a more systematic manner, the mortality did not exceed 4 per cent. Leube has recently shown that out of 556 cases which came under his care and were treated in a thorough manner, 74 per cent. were cured, 22 per cent. were much relieved, 1.6 per cent, remained in the same state, and 2.4 per cent, died. We may therefore conclude that when the disease is taken in hand at an early stage and treated in a systematic fashion, the mortality does not exceed 4 per cent.

Although it is probable that about 75 per cent. of the cases of ulcer of the stomach undergo cicatrisation, the disease is

accompanied by so many dangers, whose onset it is impossible either to predict or to prevent, that the greatest caution must always be exercised in formulating an opinion as to the progress and termination of any individual case. The difficulty of prognosis is also increased by the fact that the sequelæ of ulceration are often more serious than its immediate effects, for a large proportion of the cases that are described as 'cured' eventually succumb to malnutrition arising from cicatricial deformities of the stomach, to hypersecretion, recurrent ulceration in the scar, or to carcinoma. Each case must therefore be judged upon its own merits, special attention being directed to the age of the patient, the length of time the disease has existed, and the possibility of enforcing a systematic method of treatment.

The greatest danger to life exists between the first and fifth years of the disease, when the ulcer is especially apt to erode a large blood-vessel or to perforate the coats of the stomach.

Hæmorrhage may prove fatal either from a sudden and excessive loss of blood or from the general exhaustion which follows repeated attacks of moderate severity. Out of our seventeen cases of fatal hæmorrhage, eleven were stated to have died immediately, while in the remaining six death occurred from progressive asthenia within two to six weeks after the initial attack. These figures do not include the numerous cases in which the loss of blood seemed to predispose to the development of pneumonia or some other intercurrent affection.

A first hæmorrhage is rarely fatal unless the disease is complicated by cirrhosis of the liver or enlargement of the heart, but cases are sometimes encountered where a second and fatal attack follows the initial seizure within a short time. A rapidly fatal hæmorrhage may occur quite unexpectedly, or it may be preceded for a few days by an increase of the pain or by intolerance of food. It is to be especially apprehended whenever the symptoms of ulceration suddenly recur after a long period of quiescence. Although the accident is usually accompanied by hæmatemesis, death may take place quite suddenly, without any external evidence of bleeding; indeed, in most of the cases which apparently die of syncope the stomach and intestines are found at the autopsy to be filled with blood.

When the bleeding recurs every few days the prospects of ultimate recovery are always dubious. As a rule, the state of

the pulse affords the most reliable information. As long as it does not exceed about one hundred and twelve beats per minute, and continues to present a fair degree of tension, no immediate danger need be apprehended; but a steady increase in its rapidity with diminished force is a sure sign of exhaustion, and often indicates the occurrence of internal hæmorrhage. It must be remembered, however, that cardiac exhaustion is also apt to ensue from insufficient nutrition, so that before it can be concluded that the symptoms are due to the loss of blood care must be taken to ascertain whether the rectal feeding is being carried out in an efficient manner. Among the other signs of pressing danger are great restlessness, delirium, convulsions, coma, and a sudden fall or rise of temperature.

The appearance of the vomit is of great assistance in determining the severity of the hæmorrhage. When a large vessel has been destroyed the bleeding is profuse, and the vomit consists of bright blood mixed with recent coagula. As long as this continues, the danger remains imminent; but if the ejecta gradually become darker in colour, or assume the appearance of coffeegrounds, it may be safely assumed that the bleeding-point is becoming blocked by clot, and that if circumstances continue favourable the hæmorrhage will soon cease. It is needless to add that in all cases of ulcer the stools must be examined with the same care as the vomit, and that any looseness of the bowels attended with altered blood in the evacuations should be regarded with the greatest suspicion.

Perforation of the stomach may occur at any stage of the complaint, but it is most frequent between the second and fourth years. The earlier the accident takes place the greater is the probability that the ulcer is situated upon the anterior wall, and the greater therefore the chance of recovery if an immediate operation is undertaken. When perforation occurs after the disease has existed for many years, it is usually caused either by a recent extension of the ulcer or by rupture of the adhesions around its base.

Perforation with general peritonitis is so uniformly fatal that it may be stated at once that in the absence of surgical interference death will certainly ensue within a few days. It is true that Blume, Pariser, and others have collected a large number of cases in which recovery took place under medical treatment; but in the absence of post-mortem evidence

it is open to doubt whether the symptoms were not due to local

rather than to general peritonitis (p. 145).

The state of the digestion is always an important element in the prognosis. As long as the pain after food is moderate in degree and vomiting is infrequent, and especially if these symptoms are relieved by careful dieting, there is a good prospect of ultimate recovery if no complication occurs. On the other hand, a steady increase of pain with intolerance of milk is a sign of evil omen. When relapses occur at intervals the prognosis is very uncertain, owing to the liability to perforation and hæmorrhage.

A gradual loss of flesh and progressive anæmia are usually late symptoms of the disease, and almost always forebode a fatal termination. In such cases the sulphocyanide of potassium in the saliva becomes greatly diminished in amount, and its total disappearance is usually followed by death within a month.

The condition of the gastric secretion exercises an important influence upon the course of the disease. When simple hyperacidity accompanies the ulcer the prospect of recovery is not materially affected; but when there is a continuous hypersecretion the probability of a cure becomes remote.

The various sequelæ of the complaint greatly add to its danger. Foremost among them must be reckoned localised abscess beneath the diaphragm, which, unless promptly and efficiently treated, usually terminates fatally within a few weeks. Attacks of tetany are also extremely dangerous, while the various deformities of the stomach that ensue from the cicatrisation of the sore exert a most deleterious effect upon the health of the patient.

CHAPTER IV

THE DIAGNOSIS OF CHRONIC GASTRIC ULCER

When a patient has suffered for a length of time from severe pain after meals, with vomiting which relieves the pain, occasional attacks of hæmatemesis, and local tenderness over the epigastrium, the diagnosis of chronic ulcer of the stomach is extremely simple; but if one or more of these cardinal symptoms happens to be absent, an accurate diagnosis is always difficult, and may be impossible. For the purpose of differential diagnosis it is therefore necessary to consider each of the clinical varieties of the disease as a separate entity (p. 215).

- (1) The Gastralgic Form.—When intense pain constitutes the principal symptom of gastric ulcer the disease may be confounded with hyperacidity, hypersecretion, hyperæsthesia of the stomach, nervous gastralgia, biliary colic, and certain affections of the spinal cord.
- (a) The symptoms of hyperacidity differ from those of ulcer in the following particulars: The pain which is excited by the ingestion of food does not usually develop until one or two hours after the meal. It commences as a burning sensation in the epigastrium, which gradually increases in intensity and may extend over the whole of the abdomen and chest. At the crisis of the attack a cramping pain is experienced behind the sternum and in the throat, followed by regurgitations of an extremely acid fluid. These symptoms are temporarily relieved by draughts of warm water or milk, and removed by a full dose of bicarbonate of sodium. At the height of the attack the epigastrium may be somewhat tender, but the localised pain of ulcer is absent. Vomiting is rare, hæmatemesis never occurs, and the stomach is found to be empty in the early morning.

(b) The distinction between hypersecretion and chronic ulcer of the stomach is much more difficult, owing to the

frequent coexistence of the two complaints. It may be observed, however, that the former disorder commences with the symptoms of hyperacidity, which are gradually replaced by those which characterise a continuous secretion of gastric juice. Pain is not usually developed for an hour or more after meals, and is often most severe during the night or at other times when the stomach is free from food. It is also temporarily relieved by a glass of milk. Localised tenderness in the epigastrium is absent, and with the progress of the complaint the stomach becomes dilated. Vomiting is a frequent symptom, especially at night-time, when the ejecta consist almost entirely of bile-stained gastric juice, containing a slight excess of hydrochloric acid. Hæmatemesis never occurs unless the disease is complicated with ulceration. Examination of the stomach in the early morning shows that it contains a considerable amount of acid fluid. A milk diet has comparatively little effect upon the symptoms, and the disease, when well established, is seldom susceptible of a permanent cure without surgical aid.

- (c) Hyperasthesia of the stomach is more often confused with acute gastric ulcer than with the chronic form of the disease, since it is almost entirely met with in young women who are the subjects of profound anemia. In this complaint pain ensues immediately after the ingestion both of solids and liquids, and is referred to the left hypochondrium and lower part of the chest, as well as to the epigastrium. The affected parts are often painful on pressure, but the localised tenderness of ulceration is absent. In severe cases vomiting occurs immediately after food, but does not afford much relief to the pain. Hæmatemesis is invariably absent. A milk diet often aggravates the symptoms, and gastric sedatives are useless. On the other hand, a course of iron combined with aperients is followed by immediate improvement. The contents of the stomach after a test meal show a marked diminution in the amount of hydrochloric acid.
- (d) Nervous gastralgia is particularly apt to be mistaken for gastric ulcer when the painful crises terminate in vomiting. It may usually be observed, however, that the attacks come on at irregular intervals, and are not directly excited by the introduction of food into the stomach. The pain is often excruciating, but instead of radiating from one spot in the

epigastrium, it is diffused over the whole of the upper part of the abdomen, chest, and back. The abdomen is somewhat retracted during the paroxysm, and firm pressure often affords relief. Hæmatemesis never occurs. The complaint is most common in young adults or persons of middle age, and is often associated with symptoms of hysteria or neurasthenia. Rest in bed and a milk diet have little or no effect upon the disease, but forced feeding, with a course of tonic and electrical treatment, is followed by material improvement. The contents of the stomach after a test meal seldom present any excess of hydrochloric acid.

(e) In the majority of cases it is quite easy to distinguish between biliary colic and gastric ulcer, since in the former condition the symptoms only appear at intervals, and occur independently of the meals. The pain which ensues from the passage of a stone down the bile ducts is also far more severe than that of ulceration, and commences in an abrupt manner instead of gradually attaining a maximum. Its chief site is the right hypochondrium and umbilical region; and although there is usually tenderness over the situation of the gall-bladder, pressure upon other parts of the abdomen often affords relief. Severe vomiting and retching frequently occur during the paroxysm, but no amelioration of the pain ensues from the evacuation of the stomach. Rigors and pyrexia are common symptoms, and the attack is apt to be followed by jaundice; but hæmatemesis never occurs.

Cases are occasionally encountered, however, in which the symptoms of gall-stones so closely simulate those of gastric ulcer that a differential diagnosis is almost impossible.

Case XXXVI. A lady, 39 years of age, consulted us on account of severe dyspepsia, from which she had suffered for about two years. The history which she gave of her complaint was somewhat as follows: At the commencement, the principal symptoms were discomfort after food, with flatulence, acidity, and constipation; but after a short time the pain became more severe, and was followed by vomiting. For nearly eighteen months she had been unable to take any solid food, and found that even farinaceous substances disagreed. Latterly lavage had been performed each day with great relief. A few months after the illness began she had vomited some dark blood, but this had not recurred. She had lost several stones in weight, and had become very anæmic and debilitated.

On examination the stomach was found to be somewhat dilated, and there was a tender spot in the epigastrium slightly to the right of the median line. The other organs were healthy. At a subsequent period a sense of resistance was detected in the neighbourhood of the tender spot, which finally developed into a round but ill-defined tumour, which varied in size from time to time, and occasionally disappeared completely. There was never any jaundice. Notwithstanding complete rest and the most careful feeding, the patient gradually became weaker, and eventually died quite suddenly from syncope.

Autopsy.—The stomach was somewhat dilated, but there was no ulceration or other signs of disease. The gall-bladder, which was the size of a large orange, was globular in shape, and contained a considerable quantity of viscid bile, with one or two small stones. A large oval calculus was fixed in its neck in such a manner as to partially obstruct the cystic duct. The other organs of the body were

quite healthy.

It will be remarked that the above case presented most of the symptoms of gastric ulcer, viz. severe pain after food, vomiting, hæmatemesis, and localised tenderness in the epigastrium. The only sign which pointed to the existence of a gallstone was the presence of an ill-defined tumour to the right of the median line; but even this could only be detected at intervals, and did not exhibit the usual shape of a distended gallbladder.

- (f) Certain diseases of the spinal cord, such as locomotor ataxia, disseminated sclerosis, and subacute myelitis, are accompanied by severe attacks of abdominal pain and vomiting very similar to those which result from ulceration of the stomach. It may usually be observed, however, that these symptoms appear independently of the meals, and frequently occur at times when the stomach is empty. The pain affects the whole of the upper part of the abdomen, and is never referred to one spot in the epigastrium. The act of vomiting affords no relief, and hæmatemesis is extremely rare. Finally, in cases of tabes, the knee jerks are absent, the gait is ataxic, and the pupils do not react to light. (Table 20.)
- (2) The Catarrhal or Vomiting Form.—Gastric ulcer accompanied by excessive vomiting may be confused with

¹ Vulpian mentions a case in which vomiting of blood was responsible for an erroneous diagnosis of gastric ulcer.

acute catarrh of the stomach, hysteria, and the uncontrollable vomiting of pregnancy.

Table 20.—Diagnosis of Chronic Ulcer from other Painful Affections of the Stomach

	Chrouic ulcer	Hyper- acidity	Hyper- secretion	Gastrie hyper- æsthesia	Gastralgia	Gall-stones
Sex and age Pain:	30-50 years		30-50 years	Anæmie women 15-30	18-40 years	Over 25 years
Onset	10-30 min- utes after solid food	1-2 hours after all food	Between mcals and at night	Immediately after food or liquids	Irregular : in- dependent of food	Irregular and sudden
Position	Epigastrium and back	Whole of epigas- trium	Epigastrium	L. hypochon- drium and epigastrium	Whole of	Right hypo- chondrium and umbili- cal region
Tender- ness	Localised in epigas- trium	Slight, dif- fused	Usually abscut	General over apper abdo- men	Pressure re- lieves pain	Pressure re- lieves pain
Vomiting	Occasional, relieves pain	Rare	Frequent at night; ejecta bile-stained gastrie juice	Occasional; relief to pain slight	Absent	Severe retch- ing: no relief to pain
Hæmat- eniesis	Frequent and severe	Absent	Rare	Absent	Absent	Absent
Effective treat- ment	Rest, milk diet, Carls- bad salts	Milk diet, alkalies	Lavage, alka- lies, bismuth	Iron, purgatives	Sedatives, tonies	Diet, Carls- bad salts

(a) It has already been shown (p. 220) that in the absence of a definite history it may be impossible to distinguish an ulcer of the stomach accompanied by gastritis from recurrent catarrh of the gastric mucous membrane. It is to be observed, however, that the latter complaint is usually encountered in early life, and is often associated with an hereditary tendency to migraine or 'bilious headaches.' The attacks themselves appear at more or less definite intervals, and are ushered in by a sense of malaise, loss of appetite, and headache. Abdominal pain is seldom a subject of complaint, and although the epigastrium may be somewhat tender on pressure, the characteristic sore spot of ulcer is invariably absent. Hæmatemesis is never observed, and between the attacks there are no symptoms of disordered digestion. The contents of the stomach after a test meal do not contain an excess of hydrochloric acid. In chronic ulcer, on the other hand, careful inquiry will almost always elicit a history of epigastric pain at some previous period; and although there may be no complaint at the time of the attack it is usually possible to detect circumscribed tenderness in the epigastrium. The attacks themselves exhibit no special periodicity of recurrence, and usually last longer and are far

more severe than those of simple catarrh. In the intervals of apparent health the presence of hypersecretion can usually be ascertained.

- (b) Hysterical vomiting is practically confined to young and neurotic women who are the subjects of anamia. The ingestion of food does not give rise to actual pain, but solids seem to be arrested in the œsophagus. The emesis is more readily excited by liquids than solids, and is seldom preceded by nausea or retching. It occurs almost immediately after swallowing, and often before the food has had time to reach the stomach. The attacks appear at irregular intervals, and may persist for several days or even weeks. They are particularly apt to ensue after mental or physical overstrain, and to follow an outburst of an emotional nature. Notwithstanding the constant sickness the patient does not lose flesh, nor is there usually any serious impairment of the general health. Hæmatemesis never occurs. The effective treatment consists in the exhibition of tonics, forced feeding, and rest in bed.
- (c) Excessive vomiting associated with dyspepsia is sometimes observed during the early months of pregnancy, and, if the uterine condition happens to be overlooked, it is apt to be mistaken for organic disease of the stomach. In most cases. however, the patient suffers from retching in the early morning, with vomiting of alkaline mucus, whereas in chronic ulcer the ejecta under similar circumstances consist of an acid and bile-stained fluid. In the former case, pain after meals is usually absent, and the act of emesis, which occurs immediately after any kind of nourishment, is accompanied by much nausea and straining. Hamatemesis is never observed. When the intolerance of food is persistent, the patient rapidly loses flesh and strength, and, if unrelieved, may eventually fall into a typhoid state. The dietetic and medicinal treatment of ulcer is useless, and evacuation of the uterus is often the only measure which offers any chance of success.
- (3) The Dyspeptic Form.—This form of gastric ulcer may simulate two other diseases of the stomach, viz. chronic gastritis and nervous dyspepsia.
- (a) Chronic yastritis occurs about the same period of life as chronic ulcer, and is also common in those who have indulged to excess in alcohol and rich foods. Both complaints are accompanied by discomfort after meals, occasional vomiting,

tenderness of the epigastrium, and constinution. The chief points of distinction between them are as follows: (1) In gastritis the symptoms usually develop gradually and uniformly, but in the dyspeptic form of ulcer there is almost always a history of severe paroxysmal pain after food at some previous period. (2) In the former complaint the sense of discomfort is equally common after liquids and solids, and usually comes on from one to two hours after the meal; but in the latter oppression at the chest is chiefly caused by solid food, and occurs soon after its ingestion. (3) Distension of the abdomen and flatulent eructations are almost invariable in gastritis, while in ulcer, acidity is the most prominent source of complaint. (4) Vomiting in the early morning, with the rejection of stringy alkaline mucus, is a characteristic symptom of gastritis, but is usually absent in cases of ulcer. (5) The appetite in gastritis is diminished, thirst is increased, the bowels are irregular, the tongue foul, and the urine loaded with urates. In gastric ulcer the appetite is usually unimpaired, the tongue clean and red, the bowels very confined, and the urine pale, abundant, and neutral or only slightly acid. (6) Hæmatemesis occurs at a comparatively early stage of ulceration, but is seldom met with in gastritis unless the liver is enlarged and cirrhotic. (7) In gastritis the stomach is somewhat dilated, and the whole of the epigastrium tender on pressure, but the localised painful area met with in ulceration is absent. (8) After a test meal the contents of the stomach are usually devoid of free hydrochloric acid when gastritis is present, while in cases of chronic ulcer the acid is generally in excess. (9) A milk diet seldom agrees in cases of gastritis, and often increases the epigastric discomfort and vomiting, while the symptoms of ulcer rapidly improve with rest in bed and fluid nourishment.

It is only in very exceptional cases that gastric ulcer can be confused with nervous dyspepsia. This latter complaint is chiefly encountered in persons of a highly neurotic disposition, and in those who suffer from neurasthenia or other functional disorders of the nervous system. Pain in the abdomen is rarely experienced, but a sickening sense of emptiness or depression makes itself felt at the epigastrium shortly after food or during the intervals of digestion. The bowels are confined, and great prostration often follows each evacuation. Vomiting is rare, hæmatemesis never occurs, and

localised tenderness below the tip of the ensiform cartilage is absent. The appetite is diminished, and headache, inability to sleep, profuse perspirations, and general debility are frequent subjects of complaint. The contents of the stomach after a test meal seldom show an excess of hydrochloric acid. In doubtful cases the patient should be kept in bed, and restricted to a milk diet for ten days or a fortnight, at the end of which time, if there is no substantial improvement, the possibility of gastric ulceration may be safely excluded.

Table 21. -Showing the Principal Points of Distinction between Gastric Ulcer, Chronic Gastritis, and Nervous Dyspepsia

Symptoms	Gastric ulcer	Chronic gastritis	Nervous dyspepsia
Pain	Localised; 10-30 minutes after food, especially solids	Discomfort 1-2 hours after liquid and solid food	Discomfort, irregular in appearance, often when stomach empty
Acidity Flatulence .	Often excessive during digestion Occasional	Moderate; 1-3 hours after meals Constant; after food excessive	Waterbrash; rarely acidity Occasional
Vomiting	Only after solid	In early morning and after meals	Rare
Hæmatemesis .	Frequent	Only when cirrhosis of liver is present	Absent
Appetite	Good	Diminished, especially for break- fast	Variable
Tongue	Clean and red	Furred, flabby, or foul	Pointed and red or slightly furred
Bowels	Confined	Constipation, alternating with diarrhea; piles	Confined; often exhaustion after
Physical signs .	Local tenderness in epigastrium. Excess of HCl	Stomach dilated; liver enlarged; HCl diminished	Stomach often dilated; no ten- derness; HCl diminished

- (4) The Hæmorrhagic Form.—In addition to ulcer there are two other diseases which are sometimes accompanied by repeated attacks of hæmatemesis, viz. cirrhosis of the liver and thoracic aneurism.
- (a) In hepatic cirrhosis the bleeding is usually the result of rupture of a varicose vein in the region of the cardiac orifice. In almost every instance there is a history of long-continued intemperance, with retching in the early morning, loss of appetite, flatulence and discomfort after meals, an irregular

action of the bowels, and piles. As a rule the liver is enlarged, and the left lobe projects into the epigastrium and is tender on pressure, but the painful spot of ulcer is absent. There may be some ascites. The hæmatemesis is profuse, and the blood dark in colour. By means of careful dieting and full doses of Carlsbad salts, the hæmorrhage is usually controlled, and in early cases may not be repeated if the use of alcohol is abandoned.

- (b) Certain cases of thoracic aneurism are accompanied by repeated attacks of hamatemesis and melana from implication of the osophagus. As a rule, however, the symptoms of dysphagia precede the vomiting of blood for several weeks, and pain in the back, inequality of the pupils and of the radial pulses, or other signs of the disease are present to assist the diagnosis, while pain after food, vomiting, and tenderness in the epigastrium are absent (see case 2).
- (5) The Cachectic Form.—When chronic ulcer of the stomach is accompanied by emaciation, debility, and cachexia, there may be considerable difficulty in distinguishing it from cancer of the organ and pernicious anemia.
- (a) The chief points of distinction between cancer and ulcer are as follows: (1) In cancer the constitutional symptoms, such as debility and loss of flesh, usually precede the local phenomena, while in ulcer they seldom appear except at a late stage of the disease. (2) In the former the epigastric pain is lancinating in character, is often constant, and is less dependent upon the nature of the food. (3) Nausea is most frequent in cancer, and retching and vomiting occur at all times, and often increase the pain. In ulcer, on the other hand, emesis takes place at the height of the painful crisis, and is followed by almost immediate relief. (4) Profuse hæmatemesis is rare in cancer, while the rejection of small quantities of black blood mixed with mucus is frequently observed. In ulcer the hæmorrhage occurs at infrequent intervals, but is very abundant. (5) Anorexia is an early symptom of malignant disease, and the tongue is usually foul, while in ulcer the appetite is ordinarily preserved, and the tongue is red and clean. (6) A tumour connected with the stomach is the rule in cancer, but the exception in cases of simple ulcer. (7) Free hydrochloric acid is rarely observed either in the vomit or in the contents of the stomach after a test meal in malignant

DIAGNOSIS TO DILU-CHIRURGICAL SOCIETY

disease of the stomach, but is usually present in excess in cases of chronic ulcer. (8) In cancer the sulphocyanide of potassium in the saliva rapidly diminishes, and eventually disappears altogether, while in ulcer the presence of the salt can be detected until a late stage of the disease. (9) Leucocytosis after meals is usually absent in cancer, but is always present, and may be slightly increased, in ulcer. (10) A milk diet often increases the abdominal pain and discomfort in the former complaint, and the patient loses weight and strength when restricted to a liquid diet. (11) Cancer usually runs its course within eighteen months, but ulcer may continue for many years.

(b) Pernicious anamia.—In this disease shortness of breath and anemia are the initial symptoms, while those arising from a disordered state of the digestive organs develop at a later date. Pain after food is seldom complained of, but flatulence, abdominal distension, and waterbrash are constant and troublesome symptoms. From time to time these become intensified and are accompanied by vomiting, which persists for several days, while the temperature of the body is elevated, and the urine exhibits an unusually dark colour. Hæmatemesis is never observed, nor does the epigastrium present any localised tenderness. When the disease is well advanced the skin acquires the characteristic lemon tinge, the legs and feet are cedematous, and the temperature is elevated every night. The contents of the stomach are devoid of free hydrochloric acid, and may be neutral or even alkaline in reaction. The red corpuscles of the blood are vastly diminished in number and present the characteristic changes. (Table 22.)

Determination of the Position of the Ulcer.—An ulcer situated at the lower end of the esophagus or within the stomach in the immediate vicinity of the cardiac orifice is accompanied by certain characteristic symptoms. Pain ensues immediately after swallowing, and the bolus appears to stick behind the lower end of the sternum and to pass with difficulty through an obstruction. At first these phenomena only follow the ingestion of solid food, but subsequently semisolids or even liquids give rise to discomfort. After a short time a portion of the food regurgitates in the same state in which it was swallowed, and is not infrequently streaked or mixed with bright blood. Loss of flesh, anæmia, and debility

TABLE 22.—DIFFERENTIAL DIAGNOSIS OF ULCER, CANCER, AND PERNICIOUS ANÆMIA

Symptoms	Chronic ulcer	Cancer	Permeious anæmia
Pain	Severe, paroxys- mal, epigastric, and dorsal; pro- duced by solid food, relieved by milk diet	Constant, dull, or lancin*ting; increased by food and pressure	Flatulent distension; eructations and acidity; worse after food
Vomiting	Occasional during painful crisis; relieves pain	Frequent; retching severe; no relief to pain	Severe attacks at intervals
Hæmatemesis .	Frequent, profuse	Frequent; small quantities of coffee-ground vonit	Absent
Bowels	Constipation	Constipation	Constipation, alternating with diarrhea
Tongue	Clean, moist, red	Pale, and covered with fur	Pale and flabby
Appetite	Good, unless vomiting severe	Diminished or absent; dislike to meat	Diminished
Colour	Anæmia proportionate to loss of blood	Progressive cachexia	Progressive anæmia; lemon tint
Temperature . Loss of flesh .	Not elevated Gradual	Subnormal Rapid and con- tinuous	Pyrexial attacks Usually absent
Physical signs .	Tender spot in epi- gastrium; often hypersecretion; sulphocyanide normal; slight diminution in per- centage of red corpuscles; ab- dominal tumour rare	Tumour connected with stomach, smooth or no-dular, painful, increasing in size; absence of hydrochloric acid; gradual disappearance of sulphocyanide	Hæmic bruits; some dilatation of stomach; absence of hydrochloric acid; pale blood with great dimi- nution of red corpuscles and poikilocytosis

gradually ensue, and eventually the patient is only able to take mouthfuls of liquid nourishment with great difficulty. The passage of a soft tube gives rise to pain behind the ensiform cartilage, and its point is arrested about sixteen inches from the teeth. Dilatation of the œsophagus above the ulcer may sometimes be shown to exist, and severe attacks of hæmatemesis are apt to occur at intervals. The diagnosis of the disease from spasmodic stricture and carcinoma will be discussed when the various deformities of the stomach arising from ulceration are described (Part IV. chap. 2).

An ulcer on the lesser curvature is usually accompanied by

pain, which ensues immediately after solid food, and by local tenderness just below the tip of the ensiform cartilage. There are repeated attacks of hæmatemesis, and if perforation takes place a localised abscess below the left wing of the diaphragm is a common result.

Ulceration of the anterior surface chiefly occurs in the pyloric region. In many instances pain after food is comparatively slight, but when it exists it is relieved by the dorsal decubitus. There is always well-marked tenderness on pressure in the centre of the epigastrium. Hæmorrhage is infrequent, but the disease is especially prone to terminate in perforation and general peritonitis.

When the posterior wall of the stomach is affected with ulceration, pain after food is always severe, and is usually relieved by the prone posture. There is local tenderness over the middle or lower half of the epigastrium, and pressure may give rise to pain in the dorsal spine. The disease pursues a very chronic course, and is often fatal from hæmorrhage. Hypersecretion is a common sequela.

In ulcer of the pylorus the tender area is situated slightly to the right of the median line, and the pain after food is often relieved by lying on the left side. Disease in this region of the stomach is very apt to produce partial stenosis of the pyloric orifice, with secondary catarrh of the gastric mucous membrane and hypersecretion. Perforation is rare, but hæmorrhages are frequent.

The diagnosis of duodenal ulcer is always difficult, and in the absence of hæmorrhage is often impossible. The chief points of distinction between it and gastric ulcer are as follows: (1) It is much more common in men than in women (10 to 1), and is usually encountered at a somewhat later age. (2) Pain after food is less severe, and appears from three to five hours after meals. It is chiefly experienced in the right hypochondrium and umbilical region. The dorsal pain of gastric ulcer is never observed. (3) Vomiting is an inconstant symptom, and when it occurs it bears no relation to the ingestion of food, and affords no relief to the pain. (4) Hæmorrhage is very frequent, and more often proves fatal than in gastric ulcer. Melæna without hæmatemesis is especially suggestive of ulceration below the pylorus. (5) Perforation is a common termination of the complaint; and if local abscess develops it is

usually situated in the region of the navel. (6) Jaundice from obstruction of the bile duct and dilatation of the stomach from stenosis of the duodenum are the most frequent sequelæ, but hypersecretion is by no means uncommon. (7) When tenderness is present it is situated in the right hypochondrium rather than in the epigastrium. (8) The disease is not infrequently associated with gall-stones.

CHAPTER V

THE TREATMENT OF CHRONIC ULCER OF THE STOMACH AND DUODENUM

In the absence of a specific remedy the main indications in the treatment of the complaint are to remove any sources of irritation, and to give the digestive organs as much rest as possible during the time required for the cicatrisation of the ulcer. This condition of rest must be physiological as well as merely physical, as it has to embody three distinct items, viz. (1) an absence of local irritation by coarse particles of food or other materials introduced through the mouth; (2) diminution of the acid in the secretion, which stimulates the process of ulceration; (3) the inhibition of the muscular movements of the stomach, which are so inimical to repair.

Theoretically, the stomach can be maintained in a state of absolute rest by prohibiting food by the mouth, and feeding the patient exclusively by the rectum; but in practice it is found that life cannot be sustained indefinitely by nutrient enemata, and that within a few weeks the patient must either be fed by the mouth or die. The question, therefore, is how to ensure the maintenance of nutrition, and at the same time to throw the minimum of strain upon the gastric functions.

Cruveilhier was the first to point out that the problem could be solved by the adoption of a liquid diet, and indicated the use of milk for this purpose, on account of its bland character and the ease with which its various constituents can be digested and absorbed. But although the principle of liquid nourishment has been universally adopted, its enforcement requires great patience and perseverance on the part of the patient. As long as he is tormented by pain, exhausted by vomiting, or frightened by the occurrence of hamorrhage, he will readily endure the monotony of a milk diet and the irksomeness of restraint; but as soon as these symptoms have subsided he is apt to forget them, and

to endeavour in every possible way to evade the restrictions imposed upon him. It may truly be said that there is no other organic disease which offers a better prospect of permanent cure by systematic treatment, and, at the same time, no other in which such treatment is more difficult to enforce.

Following the long-established practice of English physicians, Leube and Ziemssen in Germany have introduced a form of treatment under the title of a 'rest curc.' For the first two or three weeks the patient is confined to bed, and has linseed poultices or wet compresses applied to the epigastrium. If the pain or vomiting is severe, rectal feeding is instituted for two or three days, and only small pieces of ice are allowed by the mouth. During the first fortnight the diet consists entirely of milk, or of milk diluted with barley or oatmeal water, half a cupful of which is given every hour. The milk is slightly warmed, and is slowly sipped instead of being drunk at once. In the third week the patient is fed every two hours, milk puddings, poached eggs, or bread and milk being allowed; while in the fourth week the diet is further increased by the addition of pounded raw or slightly broiled meat once or twice a day. Subsequently white fish, game, sweetbreads, potatoes, and broths are permitted.

This scheme of diet has the merit of being simple and easy of application, and is well suited for cases in which the difficulty of diagnosis renders the adoption of precautionary measures imperative. But that such a method of treatment is sufficient for the cure of a gastric ulcer of long standing is a matter which is open to doubt. All clinical experience tends to show that even under the most favourable circumstances the disease requires many months for its cure, and that the mere subsidence of the pain and vomiting is no proof that cicatrisation has taken place. We are therefore in the habit of adopting a much more rigorous system than that usually advocated, and believe that the permanent benefit which usually follows more than justifies the means.

The period of treatment may be conveniently divided into

four stages.

The first period lasts for two or three weeks, according to the severity of the local symptoms, during which time the patient should maintain the recumbent posture, while local treatment is applied to the painful region of the abdomen. In ordinary cases confinement to bed is only necessary for the first ten days, after which the patient may partly dress and lie upon a couch; but when pain and vomiting are prominent features he should remain in bed for the whole time. a rule linseed poultices or moist compresses over the stomach afford most relief, and may be continued for about ten days, after which the upper part of the abdomen is merely covered with a thick layer of cotton wool or a piece of flannel. When the pain resists these measures, a small blister may be applied over the affected area, and its raw surface dusted over with half a grain of acetate of morphine; but if extreme tenderness is present, a couple of leeches often afford more immediate relief. Counter-irritation in the form of blisters or applications of the liniment of iodine or of croton oil are also of value during the convalescent period, especially if a dragging pain is experienced on exertion; while some writers advocate the use of a metal canister filled with hot water, which can be worn continuously over the affected part. A well-fitting belt is often of great service by supporting the stomach and preventing the strain upon adhesions.

In the majority of cases rest in bed combined with a limited liquid diet is sufficient to relieve the urgent symptoms; but should pain or vomiting follow the ingestion of the food, or if hæmorrhage has recently occurred, recourse should be had to rectal feeding for three or four days, and only teaspoonful doses of iced water be allowed by the mouth (see page 171). Rectal alimentation for several weeks has been strongly advocated by Donkin, but such a procedure is by no

means devoid of danger, and is seldom necessary.

In all cases milk should form the staple diet, but the form in which it is administered must be varied to suit the requirements of each case. In order to test its effect one tablespoonful diluted with a little water may be given every half-hour, and should neither pain nor sickness ensue the dose may be rapidly increased, until the patient is able to take from four to six ounces every two hours. The object in limiting the quantity of nourishment is to avoid undue distension of the stomach, and to prevent an excessive stimulation of its motorial and secretory functions. Most persons prefer the milk slightly warm, but occasionally it may be necessary to

administer it cold, or even iced. When emaciation is a prominent feature a teaspoonful of cream may be added to each meal. After the first few days the quantity of milk should be cautiously increased, so that at the end of the third week two quarts are consumed during the course of each twenty-four hours.

It sometimes happens that pure milk gives rise oppression at the chest, nausea, or acidity, or the patient exhibits such a dislike to it that the treatment is difficult to carry out. Under these circumstances an attempt should be made to render the milk more digestible or more agreeable. acidity is the principal cause of complaint, the milk may be diluted with one-third of its bulk of lime water, or a few grains of bicarbonate of sodium may be added to each dose, or it may be mixed with a suitable quantity of Vichy, Ems, or soda water. Nausea and abdominal discomfort often arise from the formation of large masses of casein in the stomach. To prevent this, the milk should be either mixed with barley, oatmeal, or rice-water, or beaten up with white of egg. Should these measures fail to render the milk more easy of digestion, skimmed milk may be tried, or whey or buttermilk with cream may be substituted for it for a few days. It is an interesting fact that sterilised milk is usually well borne if care be taken that it acquires no unpleasant taste during the process. It must be prepared at least twice a day.

Peptonised milk is of the greatest value in the treatment of gastric ulcer when other forms are difficult of digestion. Care must be taken, however, that it does not acquire the bitter taste which results from over-peptonisation; when this exists to a slight degree it may be counteracted by the addition of a little Vichy water, or some flavouring material such as vanilla. Whenever the diet consists entirely of sterilised or peptonised milk, it is wise to administer a small quantity of orange or lemon juice each day to prevent the soreness of the gums that sometimes follows an exclusive use of such foods.

The second period lasts from the end of the second or third week until the end of the second month. As his general health improves, the patient may be permitted to indulge in gentle exercise, so that at the end of the first month it is often possible for him to resume his business or occupation, provided that it does not involve any great mental or physical fatigue.

The longer he can rest, however, the better is the prospect of an ultimate cure.

During this period the diet may be cautiously increased by the addition of eggs beaten up with the milk, and milk puddings made with ground rice, tapioca, flour-ball, soft bread, or powdered biscuit. The partially digested foods devised for infants, such as those of Nestlé, Benger, Liebig, Allen & Hanbury, or Savory & Moore, are also serviceable. The meals should be taken every three hours, and should include at least two quarts of milk per diem.

If these substances agree, a trial may be made with clear soups, freshly expressed meat juice, and various beef essences and jellies. Leube has invented a 'beef solution' which is held in great repute both in Germany and in America. It can often be taken when milk disagrees, and is easily digested. A quantity of the solution corresponding to half a pound of beef should be given during the twenty-four hours, or a tablespoonful or more may be added to other soups and broths.

The third period should last from the beginning of the third until the end of the sixth month. Although milk still continues to constitute the staple diet, the patient may be permitted to indulge in bread and milk, soft bread and butter, poached or lightly boiled eggs, scraped and pounded raw meat or meat which has been very lightly broiled, chicken-cream, and boiled white fish which has been passed through a fine sieve. A small quantity of mashed potato may also be allowed if desired.

The fourth and final period should extend from the sixth to the twelfth or eighteenth month, according to the severity of the case. During this time the diet is gradually increased, until at its termination there are only a few articles of food which have to be prohibited. Boiled white fish, such as cod, whiting, sole, and haddock, sweetbread, calves' brains and feet, tripe, and lightly cooked and tender game may be employed at first, and subsequently boiled mutton, broiled chops, underdone steaks, poultry, and ham. On the other hand, the oily forms of fish, tough meat, duck, goose, veal, crab, lobster, fat, condiments, spices, pastry, green vegetables, cheese, fruits, and tea must be avoided. As a rule all alcoholic liquors should be prohibited; but if necessity arise, a little good brandy or

 $^{^{\}rm 1}$ The Leube-Rosenthal 'beef solution' can be obtained from John Bell & Co., 225 Oxford St., W.

whisky, well diluted, may be given with the meals. Wines are apt to produce acidity. In all cases the meals must be taken at regular intervals, and each mouthful of food should be well masticated before being swallowed. Exercise immediately after food must be avoided. Should pain or vomiting recur, the patient must return at once to the milk diet.

Medicinal Treatment.—Although there is no special drug which can be said to exercise a direct influence upon the disease, medicinal remedies are of the greatest value in the

treatment of the various symptoms of the complaint.

(1) Pain.—In most instances rest in bed, combined with local applications to the epigastrium and a limited milk diet, is sufficient to relieve the epigastrie pain, but if this symptom eontinues troublesome recourse must be had to sedatives. Of these the most valuable is opium, the various preparations of which have always been held in high repute. It is usually given in the form of the tincture, five to ten minims of which may be combined with an alkaline bismuth mixture, though some practitioners prefer the solid extract (gr. $\frac{1}{4}$) administered every four hours as a pill. It is to be observed, however, that both these preparations are apt to give rise to nausea, loss of appetite, and obstinate constipation. Morphine, on the other hand, exerts an equally beneficial effect without producing so much disturbance, and is therefore to be preferred in the majority of cases. The solution of the hydroehlorate or acetate, in doses of five to fifteen minims, may be advantageously given in an alkaline mixture three or four times a day about fifteen minutes before food, the dose being afterwards gradually reduced. If the pain is excessive, or if vomiting precludes its administration by the mouth, the drug may be administered by subcutaneous injection. Some authorities advocate the use of eodeine, but its sedative effects are less pronounced, while its eost is greater. Among the other preparations of opium which are of service in particular eases are the compound ipecaeuanha and kino powders, nepenthe, and ehlorodyne. It must always be remembered that persons who suffer from painful affections of the stomach readily acquire the morphine habit, so that the greatest caution must be exercised in ordering opiates. Should it be desirable to eoneeal from the patient the faet that he is taking opium, the compound soap pill may be prescribed, or morphine administered under a special name

agreed upon by the chemist and the practitioner. Among the other sedatives which are sometimes employed are cannabis indica, belladonna, cocaine, hyoscyamus, bromide of potassium, chloral, hydrocyanic acid, chloric ether, and chloroform water. Fraser has advocated bichromate of potassium (gr. $\frac{1}{10}$), but

our experience has not endorsed his praise of it.

Brinton was a great believer in astringents for the relief of the pain of gastric ulcer, and usually prescribed five grains of the compound kino powder before meals three or four times a day, while Gerhardt recommends five minims of the solution of perchloride of iron diluted with water for a similar purpose. The latter remedy, however, has not met with much success in the hands of other physicians, while it is possible that much of the benefit which resulted from the kino powder was due to the opium it contains.

Nitrate of silver was formerly introduced with the view of stimulating the surface of a callous ulcer; but although it is extremely doubtful if such a result is ever obtained, there is no doubt that the drug is often of great service in relieving the epigastric pain. It probably acts by producing a film of coagulated albumen over the inflamed and sensitive mucous membrane round the open sore. Most writers employ the nitrate in pill form in doses of a quarter to half a grain, but Boas prefers a solution in distilled water. The dose should be gradually increased until one grain or more can be tolerated three times a day. The drug should be given before meals, and should be discontinued every fortnight for a few days, lest symptoms of argyria develop. Personally, we always use the oxide of silver in the form of a pill, commencing with half a grain and cautiously increasing it to one grain, with the necessary intermissions. The silver salts are particularly valuable when hypersecretion accompanies the organic disease.

The insoluble preparations of bismuth have long enjoyed a high reputation in the treatment of painful diseases of the stomach, and are of special value in cases of chronic ulcer accompanied by pain and vomiting. The subnitrate is the preparation usually employed, but it has the disadvantage of sometimes being acid, which renders it incompatible with alkaline carbonates. The carbonate of bismuth is equally efficacious, and moreover has the merit of neutralising to some extent the excessive acidity of the gastric contents. It is

better to avoid the use of mucilage in the mixture, as it is liable to undergo fermentation.

The usual custom is to prescribe ten to fifteen grains of the subnitrate or carbonate of bismuth with an equal quantity of bicarbonate of sodium and a few drops of a solution of morphine shortly before meals, and in the majority of cases the dose is sufficient to afford the requisite relief. Occasionally, however, a much larger amount of the bismuth is required, and Fleiner has shown that two or three drachms may be given at a time without any danger of toxic symptoms. plan which he recommends for its administration has been generally approved by Continental authorities, and is shortly as follows: The probable situation of the ulcer in the stomach having been determined, the viscus is washed out in the early morning with warm water containing a small quantity of bicarbonate of sodium. From 150 to 300 grains of the subnitrate or carbonate of bismuth are then suspended in half a pint of water, and the mixture is poured into the stomach through the tube, while the patient assumes the posture which is most likely to bring the drug into direct contact with the ulcer (page 239). In about ten minutes the salt becomes deposited upon the inner surface of the stomach, when the supernatant fluid is siphoned off and the tube withdrawn. The patient remains quiet for half an hour, and then partakes of breakfast. The procedure is repeated each morning for about ten days, and subsequently every other day. method of treatment has been highly eulogised by Matthes, Krämer, and other Continental physicians, who state that the direct application of bismuth to the ulcer not only relieves the symptoms of the disease, but aids the process of cicatrisation. No symptoms of poisoning have been observed, even after doses of six drachms, nor does constipation appear to result from this wholesale administration of the drug.

Owing to the dangers which accompany the use of the stomach-tube in cases of gastric ulcer, we prefer to administer the salt by the mouth. About one hour before breakfast the patient assumes the decubitus requisite to bring the powder into contact with the ulcer, and then quickly swallows about eight ounces of warm water in which from two to three drachms of carbonate of bismuth have been suspended. He remains quiet for one hour, and then partakes of breakfast.

This modification of Fleiner's method we have frequently employed with conspicuous success, and have never observed any injurious results from the large doses of bismuth. For the first fortnight the dose is given each morning; during the next fortnight on alternate days; and subsequently only once or twice a week.

Whatever drug is employed for the purpose of relieving the pain, it is necessary to add some alkali to it, in order to neutralise the excessive acidity of the gastric contents. In most cases the bicarbonate of sodium, in doses of fifteen to twenty grains, answers every purpose; but occasionally the solution of potash or the carbonate of lime or magnesia may be preferred. Debove and Bouveret recommend the daily administration of 300 to 400 grains of bicarbonate of sodium in divided doses, with the object of controlling the hyperacidity, which they consider to be the exciting cause of the ulcer, but this practice is apt to produce thirst, and not infrequently excites diarrhea.

In very chronic cases, where the pain is experienced one or two hours after a meal, owing to gaseous distension of the stomach, the addition of an antiseptic to the alkaline bismuth mixture will be found of the greatest value. For this purpose carbolic acid is to be preferred, and may either be given in the form of the glycerine preparation (8 to 12 minims) or as pure phenol. Resorcine (grs. 15 to 20), salicylic acid (grs. 10), salicylate of bismuth (grs. 20), and the liquor hydrargyri perchloridi (1 drachm) are also of use. If intestinal flatulence is the chief cause of complaint, creosote, guaiacol, or salol is to be preferred.

(2) Vomiting.—This symptom usually subsides after a few days' rest in bed with a liquid diet. Should it persist, a mixture containing bicarbonate of sodium, carbonate of bismuth, with a few drops of dilute hydrocyanic acid and acetate of morphine, may be given before meals. In obstinate cases it may be necessary to prohibit all food by the mouth, and to nourish the patient for a few days by nutrient enemata.

Neurotic women sometimes continue to vomit for some time after the pain and other symptoms of the disease have subsided. In such cases reliance must be placed upon nervine tonics and sedatives, combined with counter-irritation to the epigastrium. In rare instances recourse should be had to

forcible feeding (gavage) by means of a catheter passed through the nose—It is an interesting fact that the stomach will often retain food which has been forcibly injected into it, when the same nourishment given in the ordinary way is at once rejected.

When the emesis is the result of chronic gastritis, saline aperients given in the early morning, with careful dieting and the carbolic acid and bismuth mixture, almost always afford relief. In troublesome cases of this kind it may be necessary to wash out the stomach every morning before breakfast (Part

IV. chap. 3).

(3) Constipation.—This condition almost always accompanies gastric ulceration, and its proper treatment is a matter of the greatest importance. Ziemssen recommends Carlsbad water for this purpose, as it not only procures a free evacuation of the bowels, but also helps to neutralise the excess of hydrochloric acid in the stomach and prevents undue fermentation of the food. In the so-called 'rest cure' the patient takes from half to three-quarters of a pint of the water each day. In this country the Carlsbad salts are usually employed instead of the natural water. It is to be observed, however, that the natural Carlsbad salts contain an excess of sulphate of sodium, which renders them somewhat uncertain in their effect and liable to cause griping. It is therefore convenient to prescribe the artificial Carlsbad salts, according to the following formula: Chloride of sodium, 1 ounce; bicarbonate of sodium, 2 ounces; sulphate of sodium, 4 ounces. Two teaspoonfuls of this powder are dissolved in a tumblerful of warm water, and taken before breakfast each day. One or two loose evacuations should ensue during the course of the morning, and the dose of the salt may be increased or diminished according to its effect.

The Carlsbad salts are especially indicated in cases where chronic gastric catarrh or hypersecretion complicates the primary disease. Some practitioners, however, prefer the natural aperient waters of Friedrichshall, Hunyadi Janos, or Æsculap, or administer sulphate of magnesium. When salines give rise to abdominal pain or loss of appetite, the infusion of senna pods or the compound decoction of aloes may be prescribed, or small doses of cascara sagrada or compound

rhubarb pill may be given every night.

Drastic purgatives must never be used during the active

stage of the disease, and if the presence of a duodenal ulcer be suspected, even salines should be given with great caution.

The treatment of hæmorrhage and perforation has already been discussed (pages 170, 175), while that which is appropriate to the various sequelæ of the complaint will be described in Part IV.

Constitutional Treatment.—The fact that anemia interferes with the process of cicatrisation renders it important that as soon as the urgent symptoms of the complaint have subsided an effort should be made to improve the quality of the blood. For this purpose the administration of iron is of the greatest value. In most cases the blander preparations, such as the ammonio-citrate, the effervescing carbonate, tartrate, or the dialysed solution, are least apt to disagree; but occasionally the perchloride is well borne, especially after hæmorrhage has occurred. Sometimes reduced iron in the form of a pill, or the peptonate, or some natural chalybeate water is the only form in which the drug can be tolerated. In all cases the medicine should be omitted from time to time, in favour of an alkaline mixture containing rhubarb and taraxacum, while a small dose of blue pill is given once or twice a week.

It sometimes happens that iron in any form gives rise to nausea, flatulence, and loss of appetite, and under these conditions Quincke has recommended that the drug should be administered by subcutaneous injection, the preparations most suitable for the purpose being the ammonio-citrate, the dialysed solution, the pyro-phosphate, peptonate, and oleate. The solution should be recently prepared and be quite clear, and the injection must be made deeply into the muscles of the buttock or back. For our own part we are opposed to this method, as we have found that the subcutaneous injection of iron into animals is apt to give rise to severe gastro-intestinal catarrh, while Kobert has observed nephritis to ensue from a similar cause.

Among the various substitutes for iron which have been tried with variable success, manganese is probably the most valuable. It may be given in the form either of the pure dioxide (grs. 2 to 10) or of the phosphate (grs. 5).

The sulphate or valerianate of zinc is indicated in neurotic females who suffer from attacks of gastrodynia independently of the meals.

Arsenic is a favourite remedy with many practitioners, and is most advantageously prescribed in an alkaline solution, the dose of the drug being gradually increased. It is especially useful when the gastric complaint is accompanied by cachexia or symptoms of neurasthenia, and is also of value in relieving the constant pain which sometimes accompanies the disease in persons who have suffered from malaria. In these latter cases a course of arsenic and quinine will often afford more speedy relief than large doses of bismuth or morphine.

Whenever there is a history of syphilis it is wise to combine a small dose of iodide of potassium with the other remedies; for we have had numerous cases under our care, which, after resisting every form of sedative treatment, rapidly improved as soon as antisyphilitic remedies were administered.

The fact that ulcers upon the surface of the body will heal when exposed to an atmosphere of oxygen has led to attempts being made to apply the same principle to the treatment of gastric ulcer. As it is impossible, however, to employ the stomach-tube for several hours at a time to convey oxygen to the organ, recourse has to be had to such drugs as possess considerable oxidising power. But after a careful trial with large doses of chlorate of sodium, permanganate of potassium, and peroxide of hydrogen, we have never been able to convince ourselves that any real benefit resulted from their use. In like manner, acetate of lead, sulphate of copper, arsenious acid, and iodoform, which at different times have been supposed to exert a specific effect upon the disease, have now been abandoned as useless.

Loss of flesh must be combated by a selection of appropriate articles of food, and by the administration of cod-liver oil, cream, or maltine. Inunctions of oil have sometimes been employed with success. During the period of convalescence residence in a bracing climate will be found of the greatest value; but if the disease is complicated by gastric catarrh, sea air should be avoided.

GENERAL BIBLIOGRAPHY

GASTRIC ULCER

Abercrombie, Researches on the Diseases of the Stomach and Intestinal Tract, 3rd edit. 1837.

Albers, Beobachtungen, iii.

Andral, Clinique Médicale, ii. p. 75; Nouveau Journ. de Méd., tom. 15, p. 193.

Anstett, De l'Ulcère Chronique de l'Estomac. Thèse de Strassburg, 1863.

Aufrecht, Centralbl. f. d. med. Wissen., 1882, No. 31, p. 545.

Baillie, Morbid Anatomy of the Human Body, 1818, p. 149.

Bamberger, Die Krankheiten des chylopoietischen Systems, 1864.

Banti, Lo Sperimentale, 1880, p. 168.

Bardeleben, Virchow's Archiv, v. 250.

Barker, Lancet, 1850, i. p. 776.

Barling, Brit. Med. Journ., June 15, 1895.

Barlow, Path. Soc. Trans., vol. xxxviii. p. 141.

Barlow & Wilks, London Medical Gazette, May 1845.

Bauermeister, Inaug. Dissert., Halle 1890.

Beaurieux, Essai sur la Pseudo-gastralgie. Thèse de Paris, 1879.

Beer, A., Wiener mcd. Wochenschr., 1857, p. 501.

Bell, Edinb. Med. Journ. vol. vi. p. 783.

Bennett, Clinical Lectures, Edinburgh 1858.

Bergius, Ulcus ventriculi simplex, Stockholm 1850.

Bernhof, Beitrag zur Lehre vom Magengeschwüre, Riga 1852.

Bernutz, Gaz. des Hôpitaux, June 18, 1881.

Berthold, Virchow's Jahrb., 1883, ii. p. 199.

Biach, Wiener med. Presse, xxxi. 13, 1890.

Bianchi, Gaz. degli Ospedali, March 26, 1884.

Bineaux, Arch. Gén. de Méd., 1835, tom. viii. p. 214.

Birch-Hirschfield, Lehrbuch d. path. Anat., Bd. ii. p. 837, 1877.

Blume, Journ. Amer. Mcd. Assoc., January 25, 1896.

Boas, Diagnostic und Therapie der Magenkrankheiten, 1895.

Boneti, Sepulchretum, lib. iii. obs. 25, 1700.

Bouillaud, Archiv. de Méd., i. p. 534.

Boullay, Schmidt's Jahrb., vol. lxx. p. 185.

Bouveret, Maladies de l'Estomac, 1893, p. 221.

Boettcher, Dorpat. med. Zeitsch., Bd. v. p. 148, 1874.

Brenner, Wiener med. Wochenschr., 1881, p. 1301.

Brinton, Ulcer of the Stomach, 1857; Diseases of the Stomach, 1864.

Bristowe, Path. Soc. Trans., vol. ix. p. 275.

Brown-Sequard, Comptes Rend. de la Soc. de Biol., 1851.

Bruenniche, Hospitalstidende, 1887, 3 R. V. B., p. 697.

Budd, Lectures on Diseases of the Stomach, 1855.

Caldini, Memorie di Fisica della Soc. Stat. a Modena, tom. 12, p. 2.

Cannet, Revue Médicale, tom. iv. 1825, p. 527.

Carrington, Path. Soc. Trans., vol. xxxiii. p. 130.

Cavazzani, Centralbl. f. Chirurg., 1879, p. 711.

Cazeneuve, Ulcère Simple de l'Estomac, Lille 1862.

Cérenville, Rev. Méd. de la Suisse Romande, Sept. 1885.

Chambers, London Journal of Medicine, vol. iv. p. 597.

Chaussier, Dict. des Sci. Méd., tom. xl. p. 338.

Chiari, Prag. med. Woch., 1885, No. 27; Wiener med. Wochenschr., 1876, No. 13, p. 32; Wiener med. Blätter, 1881, No. 3.

Chomel, Andral's Préc. Path. Anat., ii. 116.

Chvostek, Wien. med. Jahrb., 1883, p. 1; Jahrb. f. Kinderheilk., 1883, p. 364.

Cohnheim, Vorles. über allg. Pathol., vol. ii. p. 53.

Colombo, Annali Univ. di Med., 1877.

Comte, Medical Week, 1895, p. 457.

Concato, Giorn. Internaz. delle Scienze Med., 1879, No. 9.

Copland, Medical Dictionary, iii. p. 919.

Cornillon, Le Progrès Méd., Apr. 28, 1883.

Cossy, Arch. Gén. de Méd., Nevember 1889.

Courtial, Journal des Savants, 1688.

Crampton, Trans. College Phys. of Ireland, vol. i. p. 1.

Crisp, Lancet, August 5, 1843.

Crocq, 'Vaste Ulcère de l'Estomac,' Presse Méd. Belge, 1874.

Cruveilhier, Anatomie Pathologique du Corps Humain, tome i. Paris 1829-1835, livr. x.-xx.; tome ii. 1835-1842, livr. xxx. & xxxi.; Archives Générales de Méd., 1856.

Curtis, Med. Annals of Albany, August 1880.

Czapla, De ulcere ventriculi perforante, Berlin 1861.

Da Costa, Medical Diagnosis, 5th edit., 1881.

Daettwyler, Deut. med. Wochenschr., 1882, p. 79.

Dahlerup, 'De ulcere ventriculi perforante,' Canstatt's Journal, 1812.

Dalton, Trans. N. Y. Path. Soc., vol. i. p. 263.

Debove & Rémond, Traité des Maladies de l'Estomac, p. 255.

Debove & Renault, Ulcère de l'Estomac, p. 16.

Decker, Berl. klin. Woch., 1887, p. 369.

Demarquay, Essai de Pneumatologie Médicale, Paris 1866.

Devie, Provence Médicale, 1892.

Diekinson, Path. Soc. Trans., vol. xliv. p. 83; Clin. Soc. Trans., vol. xxvi. p. 72.

Dittrich, Prager Vierteljahrschr., vol. vii.-xiv.

Donatus, De medica historia mirabili, lib. iv. ch. 3.

Doyen, Centralbl. f. Chir., July 6, 1895; Traitement Chirurgical des Affections de l'Estomac, 1895.

Drasche, Wien. med. Wochenschr., 1854, No. 67.

Dreschfeld, 'Ulcer of Stomach,' Allbutt's System of Medicine, 1897, vol. iii. p. 517.

Duplay, 'Contusions de l'Estomac,' Arch. Gén. de Méd., September 1881.

Duval, De l'Ulcère Chronique de l'Estomac. Thèse de Paris, 1852.

Duverney, Mémoires de l'Acad. des Sciences, 27, 1704.

Ebermaier, Rust's Magazine, tom. xxvi. p. 43, 1828.

Ebstein, Arch. f. exp. Path., 1874, p. 183.

Edinger, Deut. Arch. f. klin. Mcd., Bd. 29, p. 568.

Einhorn, Diseases of the Stomach, 1896, p. 187.

Ekman, Ulcus ventriculi perforans, 1850.

Engel, Prager Vierteljahrsch., 1853, vol. xl. p. 7.

Eppinger, Prager Vierteljahrsch., vol. cxvi. p. 133.

Ewald, Lectures on Diseases of the Stomach, 1892, ii. p. 425.

Faber, Württemb. med. Correspondenzbl., 1885, No. 40.

Fehr, Ueber die amyloide Degeneration. Inaug. Dissert., Bern 1866.

Fenwick, Samuel, Diseases of the Stomach and Duodenum, 1868, p. 294; Obscure Diseases of the Abdomen, 1889, p. 119; art. 'Stomach,' Quain's Dict. of Med. ii.

Fenwick, Soltau, Dyspepsia of Phthisis, 1894, p. 10; Disorders of Digestion in Infancy and Childhood, 1897, p. 280; 'Acute Perforating Ulcer of the Stomach,' Journ. of Pathol., June 1893.

Fiedler, Sitzungsber. d. Dresdencr Vereins f. Natur. Heilkunde, 1883.

Fillenbaum, Wiener med. Wochenschr., 1875, Bd. 25, p. 49.

Finny, Brit. Med. Journ., 1886, i. p. 1102.

Fioupe, Progrès Méd., 1874.

Fleckles, Wien. med. Wochenschr., 1867, p. 165.

Fleiner, Verhandl. des XII. Kongress f. inner. Med., 1893.

Flint, New York Med. Record, 1874.

Forestus, Observat. et Curat. Med., lib. 23, obs. 33.

Fox, Wilson, Diseases of the Stomach. 1872, p. 146; Path. Soc. Trans., xix. p. 239. Gairdner, Edinb. Med. Journ., 1855, p. 80.

Galliard, L'Union Méd., February 26, 1884; Essai sur la Pathogénie de l'Ulcère Simple de l'Estomac. Thèse de Paris, 1882; Archiv. Gén. de Méd., 1886, p. 66.

Garmise, Ulcus ventriculi cum peritonitide perforativa. Inaug. Dissert., June 1879. Garnier, De l'Ulcère Simple de l'Estomac et du Duodénum, Berlin 1865.

Gerhardt, 'Zur Aetiologie u. Therapie d. runden Magengeschwürs,' Wiener med. Presse, 1868, No. 1; Deut. med. Wochenschr., 1888, No. 18.

Glässer, Berl. klin. Wochenschr., 1883, No. 51, p. 790.

Glaenecke, Arch. f. Exp. Path. u. Pharm., Bd. 17, p. 466.

Godin, Essai sur l'Ulcère de l'Estomac. Thèse de Paris, 1877.

Godivier, Recherches sur la Pathogénie et le Diagnostic de l'Ulcère Simple de l'Estomac. Thèse de Paris, 1869.

Goldenberg, Internat. klin. Rundschau, Wien, February 8, 1891.

Goodhart, Path. Soc. Trans., vol. xxxii. p. 79.

Granet, Montpellier Méd., 1877.

Graves, Clinical Lectures, ii. p. 237.

Greenfield, Path. Soc. Trans., vol. xxvi. p. 168.

Greiss, Deut. med. Wochen. 1882, i. p. 79.

Griffini & Vassale, Ziegler und Nauwerck, Bd. 3, Hft. 5, p. 425.

Grilne, Inaug. Dissert., Giessen 1892.

Grunewaldt & Schröder, Ueber den Magensaft des Monschen. Inaug. Dissert., Dorpat 1853.

Grünfeld, Schmidt's Jahrb., 1883, Bd. 198, p. 141.

Günsburg, Arch. f. phys. Heilk. xi. 3, 1852.

Gutlmann, Berl. klin. Wochenschr., 1880, p. 221.

Häberlin, Deut. Archiv f. klin. Med., 1889, p. 461.

Habershon, Observations on the Alimentary Canal, 1857; Diseases of the Abdomen, Guy's Hosp. Rep., ser. 3, vol. i. p. 109.

Hacker, V., Wiener med. Wochenschr., 1883, No. 37, p. 1112.

Haller, Opuscula Pathologica, 1755, obs. 28, p. 60.

¹ This work is always incorrectly attributed by foreign writers to a Dr. Hutchinson.

Hallin, Schmidt's Jahrb., 119, p. 37.

Hauser, Das ehronische Magengesehwür, Leipzig 1883, p. 191.

Hedenius, Upsala Läkar Förhandl., xii. 1876.

Hektoen, North Amer. Praetit., Dec. 1889.

Hemmeter, Diseases of the Stomach, 1898; Med. Record, September 11, 1897.

Heubner, Archiv f. Heilk., 1871, p. 193.

Hévin, Mém. de l'Acad. Roy. de Chirurg., 1743, tom. i. p. 349.

Hilton, Guy's Hosp. Reports, 1846, p. 332.

Honigmann, De uleeribus ventrieuli rotundis, Berlin 1862.

Hudson, Path. Soe. Trans., vol. xxxviii. p. 134.

Hugénin, Correspondenzbl. f. sehweiz. Aerzte, 1876, No. 11.

Hughes, Guy's Hosp. Reports, 1846, p. 332.

Jaccoud, Traité de Pathologie Interne, tom. ii. p. 766.

Jackson, Descriptive Catalogue of Warren Anatomical Museum, p. 448.

Jago, Med. Times and Gazette, 1872, p. 409.

Jaksch, Prager Vierteljahrsch., Bd. iii. 1844, p. 1.

Janeway, Trans. N. Y. Path. Soc., vol. ii. p. 1.

Jayle, Bullet. Soc. Anat., November 21, 1891.

Johnson, Brit. Med. Journ., 1870, p. 305.

Jones, Handfield, Path. and Clin. Observations respecting Morbid Conditions of the Stomach, 1855.

Juliusburger, Virchow's Jahrb., ii. 248, 1874.

Jitrgensen, Deut. Archiv f. klin. Med., Bd. 31, p. 441, 1882.

Kade, Reil's Arehiv, Bd. iv. p. 381.

Keating, Proc. of Path. Soc. of Philad., vol. i. p. 1.

Kernig, St. Petersb. med. Woehenschr., Aug. 9, 1880.

Key, Axel, Virehow's Jahresb. 1870, Bd. ii. p. 155.

Kjönig, Norsk Mag. f. Lægevidensk., 3. h., viii. p. 589, 1877.

Klebs, Handbuch d. path. Anat., Bd. i. p. 185, 1869.

Kleine, Ueber Blindsaebildung am Magen. Inaug. Dissert., Göttingen 1895.

Kobert, Arch. f. exp. Path. u. Pharm., Bd. 16, p. 361.

Koch & Ewald, Ewald, op. eit., i. p. 117.

Kogerer, Prag. med. Woehensehr., 1890, No. 25, p. 315.

Kolaczek, Sehmidt's Jahrb., 1897, i. p. 140.

Kollmar, Berl. klin. Woehensehr., 28, p. 119, 1891.

Koraeh, Deut. med. Woehensehr., 1880, p. 275.

Korezinski & Jaworski, Deut. med. Woehensehr., 1886, p. 829; Deut. Arch. f. klin. Med., 1891, p. 478.

Krueg, Wiener med. Woehensehr., 1875, p. 752.

Kugel, Wiener med. Presse, 1869.

Lancereaux, Gaz. des Hôpitaux, 1876, p. 298; Traité de Syphilis, p. 248.

Lange, Deutsche Klinik, 1860, p. 90.

Langerhans, Vireh. Archiv, Bd. 124, p. 373.

Larghi, Annali Univ. di Med., 1866.

Lauenstein, Deut. med. Woehenschr., 21, 36, 1895.

Laveran, Areh. de phys. Norm. et Path., 1876, p. 443.

Laveran & Teissier, Nouveaux Eléments de Path. et de Clin. Méd., vol. ii. p. 1060, 1879.

Law, Dublin Hosp. Gaz., ii. 51.

Lebert, Die Krankheiten des Magens, p. 180, 1878.

Lees, Diseases of the Stomach, p. 73.

Lefèvre, Arch. Gén. de Méd., iii. tom. 24, p. 377.

Leith, Allbutt's System of Medicine, iii. p. 443; Internat. Clinics, 1895, vol. ii. p. 11.

Légroux, Arch. Gén. de Méd., Nov. 1880.

Lenhartz, Berl. klin. Wochenschr., 1888, p. 406.

Letulle, 'Origine Infecticuse de certains Ulcères Simples de l'Estomae,' Compt. Rend., tom. 106, No. 25.

Leube, Ziemssen's Handb. d. Spec. Path. u. Therap., Bd. vii. 1878; Deut. Arch. f. klin. Med, 1878; ibid. Bd. x. p. 11; Die Krankheiten des Magens, 1878, p. 98.

Leudet, Congrès Méd. de France, 1868, p. 104.

Levenstein, Schmidt's Jahrb., vol. iii. p. 105.

Leyden, Zeit. f. klin. Med., 1880, p. 320.

Lieutaud, Hist. Anatom. Med., tom. i. obs. 74, 139, 141.

Litten, Berl. klin. Wochenschr., Dec. 6, 1880; Virch. Archiv, Bd. 67, p. 615.

Littré, Mémoires de l'Acad. des Sciences, 1704, p. 96.

Lloyd, Lancet, 1843, vol. i. p. 273.

London, Wien. med. Presse, 1876, p. 714.

Lilderitz, Berl. klin. Wochenschr., 1879, No. 33, p. 493.

Luton, Rec. des Travaux de la Soc. Méd. d'Obs., 1858, vol. i.

Luxemburg, Wien. med. Presse, 35, p. 1917.

Mancini, Lo Sperimentale, 1876.

Marchiafava, Atti dell' Accad. Med. di Roma, iii. p. 114.

Martin, Diseases of the Stomach, 1895, p. 398.

Mason, Trans. Assoc. Amer. Phys., vol. viii. p. 218, 1893.

Mathieu, Arch. Gén. de Méd., 1880.

Mattei, Deut. mcd. Zeitung, July 5, 1883.

May, Med. Times and Gazette, vol. xxxiii. 1856, p, 38.

Maydl, Ueber subphrenische Abscesse, Wien 1894.

Maygrier, Bull. Soc. Anat., 1877, p. 321.

Menzel & Porco, Wien. mcd. Wochenschr., 1869, No. 31, p. 517.

Merkel, Wien. med. Presse, 1866, vii. p. 30.

Michaelis, Berl. klin. Wochenschr., 1884, No. 25, p. 393.

Michel, Berl. klin. Wochensehr., 1870, No. 49, p. 591.

Middeldorpf, Wien. med. Wochensehr., 1860, p. 33.

Miquel, Hannoverische Zeitsch. f. praktische Heilkunde, 1864; Schmidt's Jahrb., Bd. 125, p. 65, 1864.

Mohr, Casper's Wochensehr., 1842, 16.

Moizard, Soc. Méd. des Hôpit., 1885; Gaz. Hebdom. 1885, No. 20.

Moore, Path. Soc. Trans., vol. xxxiv. p. 94.

Morin, De la Perforation de l'Estomac. Thèse de Paris, 1800.

Müller, L., Das corrosive Gesehwür im Magen und Darmkanal, Erlangen 1860; Jenaische Zeitschr., v. 1870; Memorabilien, xvii. Oct. 1872.

Murchison, Path. Soc. Trans. xvii. p. 145; ibid. xxi. p. 162; Med. Chir. Soc. Trans. vol. xli. p. 11; Edinb. Med. Surg. Journ., 1857, p. 121.

Nasse, Schmidt's Jahrb., vol. 71, p. 185.

Nauwerck, Münch. med. Wochenschr., 1895, p. 877.

Needon, Wiener med. Presse, 1869, No. 42, p. 990.

Netter. Bullet. Soc. Anat., 1894, p. 104.

Neuss, Zcit. f. klin. Med., Bd. 3, p. 1.

Newman, Lancet, 1868, vol. ii. p. 728.

Nolle, cited by Ewald, loc. cit. p. 239.

Openchowski, Archiv f. path. Anat. u. Phys., Bd. 117, Hft. 2.

Oppolzer, Wien. med. Wochenschr., 1851.

O'Rorke, Trans. N. Y. Path. Soc., vol. i. p. 241.

Orth, Arbeiten aus dem path. Instit. in Göttingen, 1893, p. 63.

Osborne, Dublin Journ. of Medicine, vol. xxvii. p. 361.

Oser, Wien. med. Blätter, 1880, No. 52.

Palgrave, Narrative of a Year's Journey through Arabia, 1865.

Panum, Virchow's Archiv, xxv. p. 491.

Papellicr, Schmidt's Jahrb., 1854, p. 304.

Pariser, Deut. med. Woehenschr., 1895, p. 468.

Parker, Langston, Stomach and its Morbid States, 1838.

Pavy, Philos. Trans. 1863, p. 161; Treatise on the Function of Digestion, 1867.

Pcacock, Path. Soc. Trans., vol. i. p. 253.

Penzoldt & Faber, Berl. klin. Woehenschr., 1882, No. 21, p. 313.

Périssé, De l'Uleère de l'Estomac. Thèse de Paris, 1876.

Pctcr, Gaz. des Hôpitaux, June 23, 1883.

Pick, Zeit. f. klin. Med., 26, p. 452, 1894.

Pignal, Thèse de Lyon, 1891.

Plange, Virchow's Archiv, vol. xviii. p. 376.

Poensgen, Das subeutane Emphysem nach Continuitätstrennungen des Digestionstractus. Inaug. Dissert., Strassb. 1879.

Potain, Gaz. Hebdom., September 12, 1856.

Powell, Path. Soc. Trans., vol. xxix. p. 133.

Quetsch, Berl. klin. Wochensehr., 1884, No. 23, p. 353.

Quincke, Deut. Arch. f. klin. Med., Bd. xx. p. 27.

Rasmussen, Hospitalstidende, 1873.

Rausch, Abhandl. aus dem Gebiete der Heilkunde, Petersb. 1823, p. 142.

Reeklinghausen, Virehow's Archiv, Bd. xxx. p. 368.

Recs, Owen, Med. Times and Gaz., April 24, 1869.

Rehn, Jahrb. f. Kinderheilk., 1874, p. 19.

Rciche, Jahrb. d. hamburg. Staatskrankenanstalt, 1890, p. 180.

Reichmann, Berl. klin. Woehenschr., March 21, 1887.

Reimer, Jahrb. f. kinderheilk., 1876, p. 289.

Reinhard, Ulcus ventrieuli simplex mit Tumoren. Inaug. Dissert., Berlin 1888.

Rémond, Gaz. des Hôpitaux, November 14, 1891.

Riegel, Zeit. f. klin. Med., Bd. xii. p. 434; Deut. med. Wochensehr., 1886, p. 929.

Rindfleisch, Lehrbuch d. path. Gewerbelehre, Leipzig 1878, p. 317.

Ritter & Hirsch, Zeit. f. klin. Med., vol. xiii. p. 446.

Robertson, Edinb. Month. Journ. Med. Science, January 1851, p. 1.

Robinson, Path. Soc. Trans., vol. iv. p. 134

Rochemont, Münch. med. Wochenschr., 41, p. 1007.

Roger, Arch. Gén. de Méd., 1862.

Rokitanski, Oesterreich. med. Jahrb., 1839, Bd. xviii p. 184.

Roll, De ulcere ventriculi perforante, Amsterdam 1851.

Rosenbach, Deut. med. Wochenschr., 1882, p. 22.

Rosenheim, Pathologie und Therapie der Krankheiten der Speiseröhre und des Magens, 1891, p. 161.

Rosenthal, Wiener med. Presse, 1878, No. 45, p. 1404.

Roth, Virchow's Archiv, Bd. 45, p. 300.

Roussel, Gaz. des Hôpitaux, September 22, 1883.

Roux, Journ. de Méd., tom. iii. p. 407.

Rydygier, Berl. klin. Wochenschr., January 16, 1882.

Sabrazés, Journ. de Méd. de Bordeaux, November 29, 1891.

Sachs, Deut. med. Wochensehr., 1892, 18, p. 442.

Sangalli, Schmidt's Jahrb., 1854, iv. 45.

Säxinger, Prag. med. Wochenschr., 1865, 1 and 2.

Schaeffer, Dcut. med. Wochenschr., 1892, No. 46, p. 1038.

Schaepfer, Virchow's Archiv, Bd. vii., p. 158.

Schenk, Observ. Med., lib. iii. sect. i. p. 210.

Scheurer, Allg. Wien. med. Zeitschr., 1869.

Scheurlen, Charité-Annalen, 14, p. 158, 1889.

Schiff, De vi motorea baseos encephali, 1845, p. 41.

Schilling, Aerztl. Intelligenzbl., January 8, 1884.

Schliep, Deut. Arch. f. klin. Med. Bd. xiii., p. 455.

Schmilinsky, Jahrb. d. hamb. Staatskrankenanst., 1896, p. 388.

Schricher, Deut. Arch. f. klin. Med., June 5, 1877.

Sedgwick, Lancet, June 15, 1867.

Shattuck, Boston Med. and Surg. Journ., vol. ciii., June 1880.

Sicherer, Württemb. Correspondenzbl. 1843, Bd. xii. p. 26.

Sicbert, Casper's Wochenschr. f. d. Heilkunde, 1842, No. 29.

Sievers, Berl. klin. Wochenschr., August 1, 1898.

Silbermann, Deut. med. Wochenschr., 1886, No. 29, p. 497.

Skoda, Allgem. Wien. mcd. Zeitschr., 1871, p. 97.

Sohlern, Berl. klin. Wochenschr., 1889, No. 13, p. 272.

Sperk, Deutsche Klinik, 1867.

Stachelhausen, Inaugural-Dissert., Würzburg 1874.

Starcke, Deutsche Klinik, 1870, No. 26, p. 237.

Steffen, Glasgow Med. Journ., 1868.

Steiner, Virchow's Jahrb., 1868, p. 125.

Stewart, North-Western Lancet, Minnesota, April 1, 1891.

Stienow, Acad. de Méd. de Belgique, xviii.

Stockton, Medical News, January 14, 1893.

Stokes, Dublin Quart. Journal, 1868, 45, p. 201.

Stoll, Deutsch. Arch. f. klin. Med., 1894, vol. liii. p. 566.

Strube, De ulcerum ventriculi diagnosi, Berlin 1861.

Sturges, Lancet, February 7, 1874.

Talamon, Bullet. Soc. Anat., 1878, p. 374.

Talma, Zeit. f. klin. Med., 1890, p. 14.

Tapie, Le Midi Médic., Toulouse, April 22, 1894.

Thierfolder, Deut. Archiv f. klin. Med., 1868, Bd. 4, p. 33.

Tillmanns, Archiv f. klin. Chirurg., 1881, Bd. 27, p. 103.

Toulmouche, Arch. Gén. dc Méd., 1869, p. 734.

Traube, Deutsche Klinik, 1861, p. 63.

Travers & Farre, Med. Chir. Soc. Trans., vol. viii. p. 231.

Trousscau, Clinique Médicale, tom. iii. p. 95, 1865.

Vanni, Lo Sperimentale, 1889.

Van Kleef, Virchow's Jahresb., 1882, Bd. ii. p. 383.

Van Swieten, Comment. in Boerhavii Aphor., tom. iii. p. 152.

Veradini, Mem. intorno l' Ulcera Semplice Rotonda e Perforante dello Stomaco, Bologna 1863.

Vierordt, Medical Diagnosis, 1891, p. 341.

Virchow, Handbuch der spec. Path. u. Therap. i. 256; Archiv, v. 362; Wien. med. Woch., 1857, p. 498; Gesammelte Abhandlungen, p. 706.

Voigtel, Handb. der pathol. Anat., 1874, tom. ii. p. 470.

Welch, 'Ulcer of the Stomach,' Pepper's System of Practical Medicine, 1885, ii. p. 482.

Wencker, cited by Licutaud, Hist. Anat.-Mcd., vol. ii. p. 32, 1767.

West, Brit. Med. Journ., 1893, i. p. 731.

Whittaker, The Clinic, January 22, 1876.

Widal & Meslay, Medical Week, March 19, 1897.

Wilks & Mozon, Lectures on Pathological Anatomy, 2nd edit., 1875.

Williams, Lancet, April 9, 1842.

Williams, Roger, Journ. Anat. and Phys. vol. xvii. p. 460.

Willigk, Prager Vierteljahrschr., vol. li. p. 19.

Witosowski, Virch. Archiv, 94, p. 542.

Wollmann, Virchow's Jahresb., 1868, Bd. ii. p. 126.

Wrany, Prager Vierteljahrschr., vols. xcv. and xcix.

Zahn, Revue Médic. de la Suisse Romandc, 1882, ii. 144.

Zenker, Berlin. klin. Wochenschr., 1882.

Ziemssen, Volkmann's Samml. klin. Vorträge, 1871, No. 15.

Zweifel, Deut. Arch. f. klin. Med., vol. xxxix. p. 349.

DUODENAL ULCER

Abererombie, Ed. Med. Journ., 1824, xxi. 6; ibid., 1835, xliv. 278.

Adams, S., Amer. Med. Times, N. Y. 1863, vi. 101.

Allehin, Trans. Path. Soc., 1887, xxxviii.

Allen, J. M., Æsculapian, Kansas City 1896, i.

Alloncle, De l'Ulcère Perforant du Duodénum. Thesis, Paris, 1883.

Arnold, Bost. M. & S. J., 1878, xcix. 406.

Ash, Med. Exam. Phila., 1843, vi. 169.

Bailey, Lond. Med. Times, 1846-7, xv. 223.

Bainbridge, Lancet, 1841, ii. 56.

Baldeschi-Oddi, At. et Rend. dell' Accad. Med. Chir. di Perugia, 1889, i. 115.

Balfour, Edin. Med. Journ., 1874, xix. 933.

Barclay, Lancet, 1871, i. 377.

Bardeleben, Virch. Arch., 1853, vi. 251.

Barker, Trans. Path. Soc., x.

Barnes, Liverpool M. and S. Repts., 1871, v. 52.

Beek, J. H., Zeitschr. f. d. Staatsarznk. Erlang, 1831.

Billroth, Wien. med. Wochenschr., 1867, xvii.

Berens, Phila. Med. Times, 1878, viii. 322.

Biggs, G. P., N. Y. Med. Rec., 1893, xliv. 825.

Biggs, H. M., N. Y. Mcd. Journ., 1890, xli. 77; xlviii. 167.

Bouchaud, Bull. Soc. Anat., 1862, xxxvii. 209.

Boueher, Bull. Soc. Anat., 1866, xli. 487.

Bouveret, Lyon Méd., 1896, lxxxi.

Bower, Perforating Ulccr of Stomach and Bowels. 12mo. Lond. 1856.

Bradbury, Lancet, 1888, ii. p. 298.

Bristowe, Path. Soc. Trans., 1870, xxi. 355.

Broussais. Sur la Duodénite Chronique. Thesis, Paris 1825.

Bryant, J. D., Sem. Méd., 1893, p. 335.

Buequoy, Arch. Générales, 1887, i. 398.

Burdick, N. Amer. Pract. 1895, vii. 98.

Cambell, Illust. Quart. M. and S. Journ., N. Y. 1882, i. 57.

Capilan, Bull. Soc. Anat., 1878, liii. 170.

Carpenter, N. Y. Med. Journ., 1885, xlii. 524.

Castiaux, Bull. Soc. Anat., 1869, xliv.

Cayley & Gould, Middlesex Hosp. Repts., 1894, p. 168.

Chauffard, Gaz. des Hôp., 1871, xliv. 373.

Chaytor-White, Brit. Med. Journ., 1892, i. 1359.

Cheyrou Lagrèze, Ulcérations Gastro-intestinales consécutives aux Oblitérations Artérielles. Thesis, Paris, 1881.

Chrostee, Allg. Wien. med. Zeit., 1882, xxvii. 533.

Clark, A., Brit. Med. Journ., 1867, i. 687.

Clark, A. P., Bost. M. and S. J., 1881, eiv. 242.

Cléroux, Union Méd. du Canada, 1888, N.S. ii. 281.

Coats, Glasgow Med. Journ., 1888, p. 518.

Coats & Gardiner, M. Glasg. Path. and Clin. Soc., 1892, iii.

Collin, II., Sur l'Uleère Simple du Duodénum. Thesis, Paris 1894.

Collins, Warren, Bost. M. and S. J., 1896, exxxiv.

Cullen, Seot. Med. Surg. Journ., July 1897.

Culter, E. G., Med. and Surg. Repts., Bost. City Hosp., 1882, p. 372.

Curling, Lond. M. Times and G., 1853, vi. 554; Med. Chir. Trans., xxv. 260; Lancet, 1866, i. 484.

Cuthbertson, Lond. M. Times and G., 1867, ii. 387.

Davidson, Canad. Lancet, 1885-6, xviii.

Dean, H. P., Brit. Med. Journ., 1894, i. 1014.

Devie, Prog. Méd., Paris 1894.

Dobroclouski, Ejened. Klin. Gaz., St. Petersb. 1886, vi. 400.

Dreschfeld, Brit. Med. Journ., 1891, ii. 1263.

Duehek, Vierteljahrschr. f. d. prakt. Heilk., Prague 1853.

Dudensing, Arch. d. Heilk., 1860, i. 184.

Dunn, L. A., Brit. Med. Journ., 1896, i. 846.

Emmert, Weekly Med. Rev., St. Louis 1888, xviii.

Falkenbach, De ulcere duodenali ehronieo, Berlin, Svo. 1863.

Fenwick, S., Morbid States of Stom. and Duod., Lond. 8vo. 1868, pp. 336.

Fenwick, Soltau, Disorders of Digestion in Childhood, 1897, p. 280.

Ferguson, Med. Rec., N. Y., 1885, xxviii. 358.

Festal, Journ. de Méd. de Bordeaux, 1895.

Fletcher, Assoc. Med. Journ., Lond. 1854, ii. 735.

Foerster, A., Württemb. med. Zeitschr., 60, ii. 162.

Foquier, Expérience, Paris 1843, xii. 257.

Fox, Lancet, 1886, i. 250.

Frank, Med. Correspondenzbl. d. Württemb. aerzt. Ver., 1856, xxvi.

Fraser, Trans. Path. and Clin. Soc., Glasg. 1884, i.

Frolich, Trans. Path. and Clin. Soc., Glasg. 1857, xxvii. 118.

Garnier, L'Uleère Simple de l'Estomae et du Duod. Thesis, Paris 1865.

Gaube, Gaz. Hebd. de Méd., Paris 1888, xxv. 25.

Geinatz, Vratch., St. Petersb. 1894, xv.

Gerard, Journ. Gén. de M., Chir. et Pharm., Paris 1804.

Gibbon, Trans. Path. Soc., vol. vi. p. 189.

Gibert, Bull. Acad. Roy. Med., Paris 1851-2, xvii. 640.

Gordon, Dub. Mcd. Journ., 1866, xli. 90.

Gray, Lancet, 1882, i. 779.

Greenwood, Lancet, 1880, ii. 298.

Gurrieri, Riv. Clin. di Bologna, 1865, iv. 357.

Habershon, Tr. Path. Soc., vol. xxvii. p. 135.

Haldane, Edin. Med. Journ., 1863, viii. 214.

Halpin, Ind. Med. Gaz., Calcutta 1882, xvii. 298.

Haman, Med. News, Phila. 1891, lix. 131.

Hawkins, Tr. Path. Soc., 1850, i. 290.

Hcbb, Westminster Hosp. Rcpts., 1891, vii. 84.

Henderson, Lancet, 1882, ii. 1060.

Henrot & Pellot, Un. Méd. du Nord-Est, Rheims 1877, i. 199.

Herapath, Lancet, 1844, i. 33.

Hewitt, P., Tr. Path. Soc., vol. ii. p. 256.

Hlava, Allg. Wien. med. Zeitg., 1882, xxvii.

Hunter, Tr. Path. Soc., vol. xli p. 105.

Hutchinson, Lancet, 1875, i. 857.

Hutton, Dub. Journ. Med. Sc., 1848, vi. 189.

Jancivay, Med. Rec., N. Y. 1885, xxvii. p. 442.

Johnstone, W. W., Amer. J. M. Sc., 1888, xevi. 43-54.

Jollye, Brit. Med. Journ., 1892, i. 67.

Kelynack, Brit. Med. Journ., 1894, ii. p. 914.

Klinger, Arch. f. phys. Heilk., Würzb. 1861, ii. 5.

Knecht, Arch. d. Heilk., 1877, xviii.

Krannhals, St. Petersb. med. Wochenschr., 1891, p. 27.

Kranss, J., Das perforirende Geschwür im Duodenum. Berlin 1865.

Lambert, Gaz. des Hôp., 1840, ii. 379.

Lange, Ann. Surg., 1893.

Lediard, Med. Times and Gaz., 1878, i. 88.

Lenepvcu, Bull. Soc. Anat., Paris 1839, xiv. 7.

Lennander, Upsala Läk. Förh., 1891, xxvi.

Le Renard, De l'Ulcère Perforant du Duodénum. Thesis, Paris 1891.

Lcwtas, Ind. Med. Gaz., Calcutta 1884, xix.

Lieberman, B. et Mém. Soc. Méd. d. Hôp., Paris 1875, 132.

Liljebjörn, Med. Zeitg., Berlin 1857, xxvi.

Lioncau, Bull. Soc. Anat., 1867, 42, 429.

Lionville, Bull. Soc. Anat., 1867, 42, 670.

Loche, Etude sur le Traitement des Affect. de l'Estom. et du Duod. Thesis, Paris, 1893.

Lockwood, Lancet, 1891, ii. 1104; Trans. Med. Soc., Lond. 1894-5.

Loomis, N. Y. Med. Rec., 1879, xv. 88.

Luke, Lancet, 1844, i. 387.

McCarthy, Lancet, 1874, i. 120.

McKcnzic, Lancet, 1888, ii. 1060.

McKenzie, H. W. G., St. Thomas's Hosp. Repts., 1892, xx. 341.

McPhedran, Canad. Pract., 1890, xv.

Markoc, Annalist, N. Y. 1847, ii. 88.

Martyn, Brit. Med. Journ., 1864, i. 375.

Mayer, A., Die Krankheiten des Zwölftingerdarms, Düsseldorf 1844; Ann. Soc. de Méd. d'Anvers, 1866, xxvii. 118.

Moore, Tr. Path. Soc., 1880, xxxi. 108; 1883, xxxiv. 98.

Morot, Essai sur l'Ulcère du Duod. Thesis, Paris 1865.

Müller, L., Das corrosive Geschwür im Magen u. Darmkanal. Erlang. 1860.

Murchison, Path. Soc. Trans., vol. ix. 197; xx. 174.

Murray, Path. Soc. Trans., vol. xli. 104.

Myers, Path. Soc. Trans., vol. xli. 101.

Nick, Med. Correspondenzbl. d. Württemb. aerzt. Ver., 1856, xxvi. 243.

Nidergang, Essai sur l'Ulcère Simple du Duod. Thesis, Paris 1881.

O'Hara, Trans. Path. Soc., Phila. 1877, vi. 37.

Olive, Gaz. Méd. de Nantes, 1885-6, iv.

Ormerod, Lancet, 1846, ii. 423.

Ory, Bull. Soc. Anat., 1873, xlviii. 180.

Osler, Med. Rec., N. Y. 1888, xxxiv. 609; Montreal Gen. Hosp. Path. Repts., 1877, i. 45; 1880, i. 306.

O'Sullivan, Dubl. Med. Journ., 1864, xxxvii. 221.

Oulmont, N., Arch. Méd. de Strassb., 1836, iii. 418.

Paget, S., Brit. Med. Journ., 1890, i., p. 148.

Peabody, Med. Rcc., N. Y. 1890, xxxvii. 453.

Pepper, J., Am. Med. Assoc., Chicago 1889, xii. 724; Mcd. and S. Repts., Phila. 1889, lxx.

Pepper & Griffith, Amer. J. M. Sc., 1888, xcv. 34.

'Perforations and Ulcers of the Duod.,' Tr. N. Y. Path. Soc. 1876, i. 245.

Perry, Trans. Path. Soc., vol. xliv. 84.

Perry & Shaw, Guy's Hosp. Repts., 1893, l. 171.

Petrequin, Arch. Gén. de Méd., 1836, xlii.

Pierson, Calif. State Med. Journ., 1856-7, i. 30.

Pileher, N. Y. Med. Journ., 1894, lix. 346.

Potain, Union Méd., Paris 1889.

Powell, N. Orleans M. and S. Journ., 1854, xi. 468.

Powles, Med. Times and Gaz., 1865, ii. 196.

Pusinelli, Berlin. klin. Wochenschr., 1887.

Ranking, Brit. Med. Journ., 1859, ii., p. 723.

Rayer, Arch. Gén. de Méd., 1825, vii. 166; Gaz. des Hôp., 1839, i. 137.

Rickman, Ulcus duod. und seine Diagnose. Thesis, Berlin 1893.

Roberts, Lond. Med. Gaz., 1845, xxxvi. 1419.

Robinson, F., Tr. Path. Soc., 1868, xix. 236.

Rodgers, Lancet, 1871, ii. 159.

Rolleston, Path. Soc. Trans., vol. xlii. 183.

Sargent, Amer. J. M. Sc., 1854, N.S. xxviii.

Schultz, B., Beitr. z. Kennt. des perfor. Duodenalgeschwürs. 8vo, Greifswald 1873.

See, G., Gaz. des Hôp., 1893, lxvi. 699.

Seigel, A., Ueb. das chronische Duodenalgeschwür. Würzburg 1877.

Shaw, Ext. Rec. Bost. Soc. M. Improv., 1880, vii. 98.

Shield, Lancet, 1895, i. 1169.

Spiegelberg, Jahrb. f. Kinderh., 1868-9, ii. 333.

Spitta, Brit. Med. Journ., 1875, i. p. 422.

Stampaechia, Boll. di Clin., Milano 1892, ix. 193.

Starcke, Deutsch. Klin., 1870, 26, p. 237.

Stewart, Path. Soc. Trans., vol. xiv. 173.

Stich, Deut. Arch. f. klin. Med., 1874, xiii.

Stilwell, Lancet, 1846, ii. 67.

Stokes, Dubl. Med. Journ., 1876, lxii. 327.

Streeter, Med. Rec., N. Y. 1884, xxv. 67.

Streeton, Midl. M. and S. Reporter, Worcester 1828-9, i. 127.

Swift, Iowa State Med. Rept., 1883, i.

Teillais, A., De l'Ulcère Simple du Duodénum. Thesis, Paris 1869.

Travers, Lond. Med. Chir. Trans., 1817, viii. p. 232.

Treibmann, Ueb. das perfor. Duodenalgeschwür. Svo. Leipzig 1867.

Trier, F., Ulcus corrosivum duodeni. 8vo, Copenhagen 1863.

'Ulcer of Duod.,' West. J. M. and Phys. Sc., Cincin. 1838, xi. 510.

' Une Perfor, du Duod.,' Journ. Univ. d. Sc. Méd. de Paris, 1828, li. 255.

Vallon, Zeitschr. d. k. Gesell. d. Aerzte zu Wien, 1854, ii.

Van Buren, N. Y. Med. Times, 1852, ii. 353.

Wadham, Lancet, 1871, i. 230.

Wagner, Arch. d. Heilk., 1864, v. 372.

Wait, Bost. M. and S. Journ., 1841, xxiv. 237.

Wallis & Svenson, Hygeia, 1888, l. 342.

Wallmann, Wien. med. Wochenschr., 1858, viii.

West, Tr. Clin. Soc., Lond. 1886, xix. 113.

Weston, N. Y. Med. Rec., 1892, xli. 525.

Wood, Therap. Gaz., Detroit 1879, v. 18.

Woods, Med. Press and Circ., Lond. 1878, N.S. XXV.

Zacchi, Lo Sperimentale, 1888, lxii. 289.

Zahn, Rev. Méd. Suisse Rom., 1882, ii. 144.

SURGICAL TREATMENT

Aitehison, Brit. Med. Journ., 1894, ii. p. 864.

Anson, Lancet, 1893, i. p. 469.

Armstrong, Montr. Med. Mag., 1896, p. 505.

Atherton, New York Med. Rec., 1895, i. p. 2.

Barker, Brit. Med. Journ., 1896, ii. p. 1583; Surgery of the Stomach, 1898.

Barling, Brit. Med. Journ., June 17, 1893.

Bennett, Lancet, 1894, ii. p. 21.

Bowlby, Birm. Med. Rev., 1895, p. 98.

Braun, Centralbl. f. Chirurg., 1897, p. 24.

Cahn, Berl. klin. Woch., 32, 28, 1895.

Campbell, Brit. Med. Journ., 1898, i. p. 150.

Carle & Fantino, Arch. f. klin. Chirurg., 56, 1, p. 1; 2, p. 217, 1898.

Cheyne, Lancet, 1895, i. p. 1253.

Comte, La Sem. Méd., 1895, p. 406.

Czerny, Archiv f. klin. Chirurg., 1888, p. 853.

Dalziel, Glasgow Med. Journ., 1896, p. 302.

Dean, Med. Soc. Trans., 1894, p. 305.

Dunn, Lancet, 1895, i. p. 1252; Brit. Med. Journ., 1896, i. p. 846.

Eve, Lancet, 1894, ii. p. 1091.

Gannet & Mixter, Boston Med. Surg. Journ., 120, 2, p. 38.

Garre, Münch. Med. Woch., 45, 37, 1898.

Gilford, Lancet, June 2, 1894; Brit. Med. Journ., 1893, i. p. 944.

Hartmann, Mercredi Méd., 1895, p. 163.

Haslam, Brit. Med. Journ., 1893, ii. p. 1044.

Haward, Brit. Med. Journ., 1893, i. p. 952.

Helfrich, Deut. Med. Woch., 1895, p. 451.

Hofmeister, Beiträge zu klin. Chirurg., 1896, ii. p. 351.

Horrocks, Lancet, 1895, ii. p. 413.

Horsley, Brit. Med. Journ., 1895, ii. p. 78.

Jowers, Lancet, 1895, i. p. 544.

Kirkpatrick, Montr. Med. Mag., 1895, xxiii. p. 670.

Kocher, Correspondenzbl. f. schweiz. Acrzte, 28, 20, 1898.

Kohler, Charité-Annalen, 1888, p. 450.

Kriege, Berl. klin. Wochen., 1892, p. 1280.

Küster, Arch. f. klin. Chirurg., 48, iv. p. 787, 1894.

Lamphear, Am. Journ. Surg. and Gynæc., Dec. 1895.

Lauenstein, Deut. med. Woch., 1895, 21, 36.

Le Dentu, Bull. de l'Acad. de Méd., 1897, 61, 18.

Leube, Arch. f. klin. Med., 55, i. p. 69, 1897.

Lockwood, Lancet, 1894, ii. p. 968.

Lücke, Centralbl. f. Chirurg., 1893, p. 28.

Lundic, Brit. Med. Journ., Jan. 26, 1895.

Maclaren, Lancet, 1894, i. p. 671.

McCosh, cited by Weir, op. cit.

Maylard, Treatise on the Surgery of the Alimentary Canal, 1896.

Mauricc, Brit. Med. Journ., 1895, ii. p. 980; Lancet, 1894, i. p. 1373.

Michaux, Rev. de Chirurg., 1894, p. 94.

Mikulicz, Volkmann's klin. Samml., 1885, p. 2310; Archiv f. klin. Chirurg., 1889,

p. 780; Archiv f. klin. Med., 55, i. p. 84, 1897.

Morris, Brit. Med. Journ., 1894, ii. p. 862.

Morse, Lancet, March 17, 1894; Brit. Med. Journ., Feb. 13, 1897.

Mouisset, Lyon Méd., 1890, p. 516.

Nicholson, Brit. Med. Journ., 1894, ii. p. 983.

O'Callaghan, Brit. Med. Journ., 1894, ii. p. 866.

Page, Lancet, 1894, i. p. 733.

Parsons, Dub. Journ. Med. Sci., 1892, p. 27.

Paul, Brit. Med. Journ., Apr. 6, 1895.

Pollard, Brit. Med. Journ., 1895, ii. p. 14.

Quénu, Mercredi Méd., 1895, p. 583.

Roughton, Brit. Med. Journ., July 9, 1898.

Schuchardt, Archiv f. klin. Chirurg., 1895, p. 615.

Silcock, Lancet, 1895, i. p. 1252.

Stelzner, Verhandl. d. deut. Ges. f. Chirurg., 1889, p. 98.

Sterling, Austral. med. Gaz., 1893, p. 281.

Sourdille, Bull. Soc. Anat., 1895, p. 301.

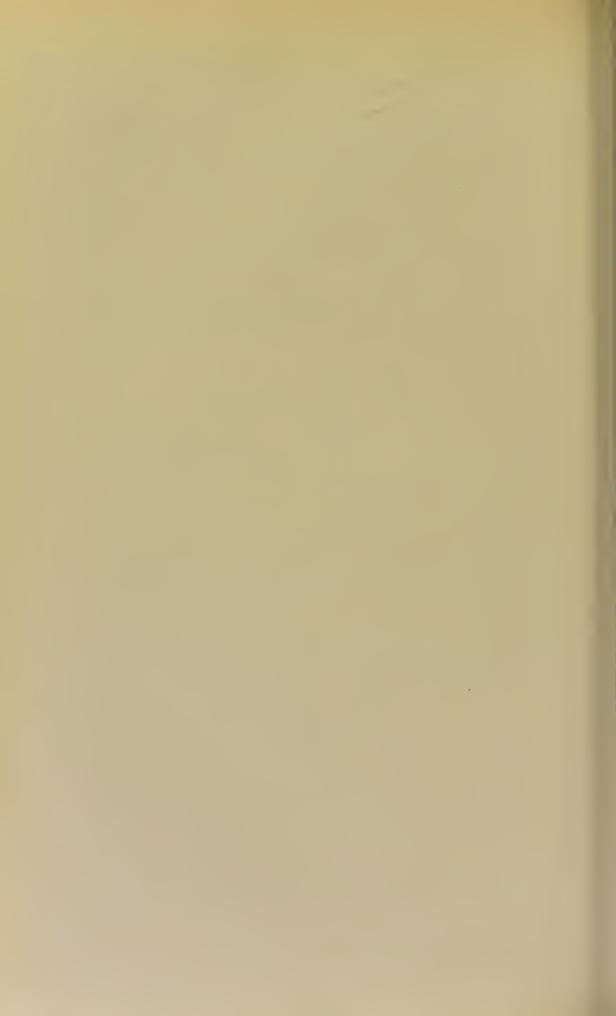
Swain, Lancet, 1194, ii. p. 21.

Taylor, Birm. med. Rev., 23, 1888.

Wahl, St. Petersb. Med. Woch., 41, 1891.

Walters, Lancet, 1895, i. p. 484.

Weir, Internat. Med. Mag., Feb. 1892.



PART IV

THE SEQUELÆ OF CHRONIC ULCER

CHAPTER I

PERIGASTRIC ADHESIONS

[To be read with pages 28-32]

THE adhesion of the base of an ulcer to some solid organ in its vicinity is such an important factor in the prevention of fatal perforation that one is apt to ignore the possibility of any ill-effects resulting from this beneficent act on the part of Nature. Clinical experience, however, seems to indicate that the presence of adhesions is often responsible for those obscure abdominal symptoms which develop after the cure of a chronic ulcer, while in not a few cases they tend to shorten life by interfering with the processes of digestion. Thus it is certain that many of the troublesome forms of 'gastralgia' which follow ulceration of the stomach are due to mechanical interference with the movements of the organ, and it is to be feared that persons are often regarded as the subjects of hysteria or hypochondriasis because the continual feeling of vague uneasiness or of 'dragging' pain in the abdomen, of which they complain, is not attributed to its proper cause. At the present time our knowledge upon the subject has been chiefly derived from post-mortem examinations, and from isolated cases where the surgeon has divided adhesions during life with immediate and lasting benefit. It is therefore only possible to indicate in a somewhat vague and unsatisfactory manner the symptoms which may be expected to attend the presence of perigastric adhesions.

(1) General Adhesions.—Diffuse plastic perigastritis is a rare result of simple ulcer, and its detection during life is extremely difficult. In the few cases which have been recorded, the patients appear to have suffered from constant pain in the upper part of the abdomen, accompanied by vomiting, con-

stipation, and gradual emaciation. As a rule, the epigastric region was distended, tender on pressure, and dull on percussion, while in a few instances there was a general sense of resistance in the region of the liver and stomach, with ascites and ædema of the legs. In one of our cases gastric symptoms were entirely absent, although the stomach was found, after death, to be so compressed as to resemble a piece of bowel. The disease is sometimes accompanied by a slight degree of pyrexia.

Adhesions around one end of the stomach which firmly bind the organ to the neighbouring viscera are usually the result of perigastric suppuration. In most of these cases the patient complains of a constant aching pain in the abdomen, which is increased by food and exercise. Acidity and flatulence are almost always present, and vomiting is not infrequent. If the colon is involved, obstinate constipation, with pain or faintness after an evacuation of the bowels, is often observed.

The physical signs of this condition are of great importance. In almost every instance where the contraction of an abscess cavity has displaced the stomach, there is an obvious lack of symmetry between the two sides of the abdomen. Thus, if the stomach has been dragged towards the left, as is usually the case, the epigastrium and the left hypochondrium exhibit an unusual degree of fulness, while the rest of the abdomen is flat or retracted. The percussion note over the swelling is hyper-resonant, and the characteristic stomach-splash may be elicited on palpation. In those rare cases where a periduodenal abscess undergoes spontaneous cure, the stomach and intestines are dragged towards the right hypochondrium and are adherent to the liver. These various conditions will be further discussed under the head of 'perigastric abscess' (Chap. V.).

(2) Adhesions between the Stomach and Liver.—When an ulcer situated near the lesser curvature has become united to the under surface of the left lobe of the liver, the chief result of the adhesion is to prevent the downward displacement and forward rotation of the stomach which normally follow the ingestion of food, and to impede the contractions of its circular and longitudinal muscular fibres. This interference with the motorial functions of the organ is probably responsible for the sense of fulness and discomfort

after meals of which complaint is so often made, and for the flatulence and acidity which ensue from the use of those articles of diet that are difficult of solution, or are prone to undergo fermentation. It is also to be observed that in many cases of this description the patient experiences a constant dragging pain in the epigastrium or right side of the chest, whenever he indulges in physical exertion after meals or suffers from anxiety or excitement. The fact that rest in the recumbent posture, or the application of an elastic belt to support the stomach, affords relief, appears to indicate that the pain is due to a mechanical strain upon the adhesions caused by the weight of the organ or by violent contractions of its walls.

The physical signs of this condition are usually ambiguous. In most instances the left lobe of the liver is slightly displaced downwards, so that its thickened margin can be felt in the upper part of the epigastrium, and in thin people can be seen to descend with the stomach on deep inspiration. If the stomach is moderately distended with gas, the mass becomes more distinct, and percussion elicits a sub-tympanitic note. Several cases of this description have come under our notice, in which the diagnosis of adhesions was confirmed by a post-mortem examination.

The results of adhesions between the pylorus and the liver are very important, and the signs which denote their existence are relatively distinct.

The first symptom to attract attention is pain in the upper part of the abdomen, which, instead of appearing soon after meals, as is usual in cases of open ulcer, occurs only towards the termination of gastric digestion. After a time the pain becomes more or less continuous, but is always aggravated by physical exertion. Instead of being referred to the epigastrium, it is located at a spot just below the margin of the right costal arch, from whence it seems to radiate over the right side of the chest, and may be felt acutely in the shoulder or scapular region. In some cases the patient complains of a tender swelling in the right hypochondrium which varies in size with the intensity of the pain.

Sooner or later vomiting supervenes. At first this symptom occurs only during a severe attack of pain, or after a meal, but it soon becomes more frequent, and often continues for several days. Occasionally the attacks of emesis alternate with the

painful seizures, but more often both symptoms are present at the same time. In severe cases the patient is never free from pain or sickness, and he consequently loses flesh and strength, and becomes extremely despondent about his condition. Unless the gastric disease is detected and treated in an appropriate manner, the emaciation becomes profound, and death finally ensues from exhaustion.

The pain and vomiting in these cases are due to obstruction of the pyloric end of the stomach arising from the attachment of the organ to the liver.

On examination of the abdomen the stomach is always found to be dilated, and its peristaltic movements are often visible through the thin abdominal wall. Pressure with the hand gives rise to pain at one spot below the right costal margin, and not infrequently a sense of resistance can be detected beneath the right lobe of the liver, which moves downwards with that organ on deep inspiration, and affords a dull note on percussion. The evanescent 'swellings,' of which the patient complains, are probably due to spasmodic contraction of the hypertrophied gastric walls. When the outlines of the stomach are mapped out with a pencil, both the lesser curvature and the pylorus are found to occupy their normal position, while the lower border of the viscus reaches several inches below the level of the umbilicus. This fact shows that the pylorus must have become adherent to the liver, for otherwise the weight of the enlarged stomach would drag it downwards and inwards, and render the line of the lesser curvature almost perpendicular.

The following cases show that the diagnosis of adhesions is of great importance in regard to treatment.

Case XXXVII. A man, 35 years of age, was admitted into the London Hospital for disease of the stomach. It appeared from his history that about five years previously he had suffered from severe pain after meals, and two years later from hæmatemesis. Latterly he had experienced constant pain in the upper part of the abdomen, and had vomited two or three times each day. He had consequently become extremely weak and emaciated. On examination the stomach was found to occupy the greater part of the abdominal cavity, and its peristaltic movements were plainly visible. The right lobe of the liver projected slightly below the costal margin, and at one spot, just to the right of the epigastrium, it presented a small ill-defined

but hard mass, which was dull on percussion and tender on pressure. The upper curvature of the stomach was in its normal position, and

apparently terminated in the region of the tuniour,

The fact that the patient had previously suffered from the symptoms of gastric ulcer, combined with the signs of great dilatation of the organ and fixation of the pylorus, rendered it probable that the base of the ulcer was adherent to the liver, and that the tumour was due to the formation of dense fibroid tissue around the point of union.

Mr. Hurry Fenwick opened the abdomen, and found that the pyloric end of the stomach was firmly attached to a small calcified hydatid of the liver. The adhesions were divided, with the result that the gastric symptoms immediately subsided, and the patient left the hospital in good health. As the stomach was not opened it was impossible to determine the site of the former ulcer.

Case XXXVIII. A man, aged 18, was admitted into the Leeds General Infirmary, under the care of Mr. Mayo Robson, who gives the following details. 'He had never had any serious illness until three vears previously, when he began to suffer now and again from severe pains, stabbing in character, situated in the right part of the hypogastrium, and lasting two or three hours. He could give no eause for the attacks, which did not always follow the taking of food.

'After he had had the pain for a few weeks, it would subside, and nausea and vomiting would follow, often lasting for six weeks. He would then be well for a month or so of both complaints; but during the whole three years he had never been free from pain or sickness

beyond a month or six weeks.

'The same sequence of symptoms usually occurred, although at times sickness accompanied the pain. On one occasion he vomited blood, and several times coffee-ground material. Three months before admission to the Infirmary the pain became localised to a spot just below the ninth rib on the right side, and he had never been a day free from pain since. He stated that a swelling could be felt at times under the right ribs when the pain was present, disappearing when the pain subsided. He complained much of offensive eruetation.

'He had never been jaundiced. Although he had picked up a little between the attacks, the loss of flesh had been progressive during the three years. When I saw him he weighed only 6 st. 2 lbs. and was suffering from profound weakness and extreme emaciation, Beyond a dilated stomach nothing else abnormal could be made out, and although there was marked tenderness under the right costal

margin, no tumour could be felt.

'A diagnosis of dilated stomach, probably dependent on adhesions of the pylorus to the gall-bladder, was made, and the patient was admitted to the Infirmary for stomach lavage and

peptonised feeding, to be followed by operation if decided relief did not occur.

'On July 26 the following note was made:—During the last fortnight, although the stomach has been washed out daily with a weak boro-glyceride solution, he has suffered as much as ever from attacks of pain, generally coming on at night, but at times during the day. On examining the abdomen during the seizures no tumour can be seen or felt. There has, however, been no vomiting since admission, and his appetite is fairly good.

'On July 30, though he had gained a little in weight, he still had the pain and was very anxious to have something done to give decided relief.

'On August 4, 1892, the abdomen having been previously asepticised, and the stomach washed out with boro-glyceride solution, ether was administered and an incision of three inches was made through the upper end of the right linea semilunaris, when adherent omentum was found covering the region of the gall-bladder like a veil; this was detached by the fingers, exposing the pylorus adherent to the gall-bladder and under surface of the liver. The adhesions were freely separated and the bleeding was arrested, chiefly by sponge pressure, two or three catgut ligatures only being required to complete the hæmostasis. The wound was then closed by three rows of sutures,—a continuous one for the serous membrane, interrupted sutures for the aponeurosis, and the same for the integuments. Recovery was uninterrupted, and the wound healed by first intention.

'On August 15 he said he was quite free from pain and felt well. He was kept recumbent for three weeks, although he was allowed to take food freely.

He returned home at the end of eight weeks, and had gained nearly 2 st. in weight.'

(3) Adhesions with the Pancreas.—Owing to the frequent coexistence of chronic gastric catarrh and hypersecretion, it is difficult to determine the exact symptoms which ensue from this morbid condition. In some of our cases, however, where an autopsy showed extensive adhesion of a healed ulcer to the pancreas, the patients had complained of constant pain in the lower dorsal spine and in the region of the navel, which rendered them unable to pursue any laborious occupation. Where the ulcer is still active the tissue of the pancreas may be gradually eroded and its secretion detected in the contents of the stomach. In very rare instances pieces of the gland become detached and may perhaps be recognised in the vomit.

Adhesion of the duodenum to the liver may produce jaundice by compression of the bile duct, or if the cystic duct is involved, dilatation of the gall bladder may result.

Treatment.—If there are no symptoms of gastric dilatation, the food should consist of substances which are easily digested, and should be given in small quantities at frequent intervals. A bandage or elastic belt applied firmly round the upper part of the abdomen, so as to support the stomach, almost always affords relief by diminishing the strain upon the adhesions. When constant pain is a troublesome feature of the case, counterirritation, in the form of blisters or iodine liniment, may be applied to the skin, or the part may be rubbed with the liniment of croton oil.

In severe cases it may be necessary to administer morphine or other sedative remedies. In almost every instance the tendency to constipation requires correction by means of castor oil, cascara, or the aloes and iron pill.

When vomiting occurs as the result of dilatation of the stomach, excess of fluid with the food must be avoided, and all articles which are prone to undergo fermentation must be excluded from the dietary. Fermentation of the gastric contents can often be controlled by the exhibition of carbolic acid and other antiseptics combined with alkalies or bismuth, but if these fail recourse should be had to lavage.

The question of surgical interference has always to be considered, since the removal of the adhesions can only be effected by operative measures.

If the signs point to the existence of extensive adhesions, while the pain can be relieved, though perhaps not entirely removed, by medical treatment, it is seldom advisable to submit the patient to an operation which is beset by many difficulties and the object of which it is often impossible to achieve. The same remark applies to those cases where the stomach is adherent to a large surface of the liver or the pancreas, although Küster is stated to have operated with success upon two cases of the latter description.

Undoubtedly surgical interference achieves its principal success where fixation of the pylorus to the liver has given rise to dilatation of the stomach, or when the stomach is adherent to the abdominal wall. In those reported by Mayo

Robson the pain and vomiting were immediately relieved by the division of the adhesions about the pylorus, and the same happy result ensued in our case (37). Even when the diagnosis is doubtful, an exploratory incision over the site of the pain would do no harm, even if further procedure was found to be impossible.

CHAPTER II

THE RESULTS OF CICATRISATION

Stenosis of the Pylorus—Stenosis of the Cardia—Hour-glass Deformity—
Obliteration of the Bile Duct
[To be read with pp. 38-49]

(1) Dilatation of the Stomach.—Of the various consequences of the cicatrisation of a gastric ulcer stenosis of the pylorus with consecutive dilatation of the stomach is the most frequent and important.

As a rule the enlargement of the viscus takes place very gradually, and its symptoms imperceptibly replace those of the primary complaint, but in old or debilitated persons it may commence quite suddenly and develop with considerable rapidity. When the pyloric obstruction is due to the action of mineral acids or other corrosive poisons upon the tissues, the disease also pursues a somewhat acute course.

Pain is seldom a prominent feature of the disease unless the ulcer remains partially unhealed or the pylorus has contracted adhesions with the liver. In almost every instance, however, the patient experiences a feeling of fulness and discomfort after meals, or complains of a burning sensation at the epigastrium during the period of digestion. The former symptom usually arises from gaseous distension of the stomach and the consequent drag of the enlarged organ upon the liver and diaphragm, while the latter owes its origin to irritation of the gastric mucosa caused by an excess of hydrochloric acid in its secretion. In rare instances a severe spasmodic pain is experienced in the region of the pylorus, owing to the violent contractions of the stomach which are necessary to force the chyme through the contracted orifice.

Vomiting is an invariable symptom of chronic dilatation of the stomach. At first it occurs only occasionally, and is usually attributed by the patient to some indiscretion in diet, but

it gradually becomes more frequent, until at length hardly a day passes without one or more attacks. In its most typical form the emesis occurs several hours after a meal or during the early part of the night. It is usually preceded by nausea, palpitation, and faintness, or by the eructation of gas and mouthfuls of a highly acid fluid. The act itself is essentially a regurgitation of the contents of the stomach, and is seldom accompanied either by retching or straining. The ejecta vary in quantity from one to seven pints, and are most copious when the vomiting occurs at comparatively rare intervals. They con-

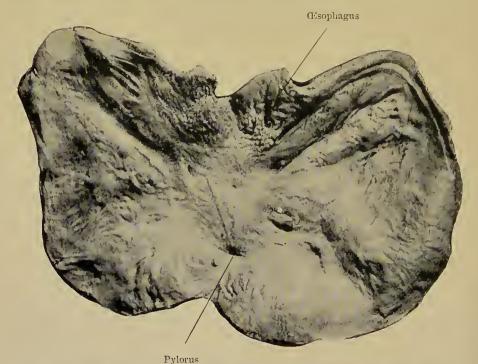


Fig. 49.—Cicatricial stenosis of the pylorus, with contraction of the stomach. (London Hospital Museum.)

sist of a turbid, brownish, sour-smelling fluid, which deposits particles of undigested food and mucus upon standing, and usually contains an excess of free hydrochloric acid. Torulæ, sarcinæ, numerous bacteria, starch granules, and pieces of vegetable matter may be recognised in the sediment when examined with the microscope.

Attacks of subacute inflammation of the gastric mucous membrane are apt to supervene from time to time, and give rise to serious symptoms. Pain is seldom a source of complaint, but retching and vomiting are incessant, and preclude the administration of any form of nourishment by the mouth.

Under these conditions the vomited matter soon loses its usual character, and consists almost entirely of alkaline mucus mixed with yellow or green bile. The attacks may last from a few hours to two or three weeks.

In all cases of gastric dilatation the patient loses the desire for food and suffers from obstinate constipation. The secretion of urine is greatly diminished and the fluid is usually neutral or alkaline in reaction. Phosphaturia, with painful or difficult micturition, is often observed. The tongue is covered with a grevish-white fur, the breath is sour and offensive, and the gaseous eructations not infrequently smell of sulphuretted hydrogen. As a rule the loss of flesh takes place gradually, but during the attacks of secondary gastric catarrh, emaciation proceeds rapidly and the patient becomes dangerously debilitated. Among the minor symptoms of the complaint are numbness of the hands and feet, dyspnæa on exertion, asthma after meals, irritation and dryness of the skin, swelling of the finger joints, attacks of giddiness and faintness, albuminuria, and ædema of the ankles. The convulsive seizures which occasionally ensue from dilatation of the stomach will be described in a separate chapter (Chap. IV.).

Physical signs.—When the abdomen is examined the natural prominence of the epigastrium is seen to be replaced by a transverse furrow or depression, while the umbilical region and the left hypochondrium are occupied by an elastic swelling which affords a splashing sound on palpation, and over the surface of which the peristaltic movements of the stomach are usually visible. The dimensions of the viscus are readily determined by distending it with gas, or by employing the method of auscultatory percussion. In most instances the posterior surface of the organ is fixed to the liver or pancreas, so that the lesser curvature, although displaced downwards, still pursues its normal direction; but if no adhesions have taken place the pylorus is drawn downwards and inwards, and the upper margin becomes almost perpendicular.

Another important sign of gastric dilatation is the presence of food in the organ in the early morning. In healthy persons only a few drachins of fluid can be withdrawn by the tube before breakfast; but when the transmission of food into the intestine is delayed, a considerable quantity of partially digested material can be evacuated

The elimination of sulphocyanide of potassium by the salivary glands is always diminished, and in long-standing

cases the salt may disappear entirely from the secretion.

Diagnosis.—Cicatricial stenosis of the pylorus is very apt to be confused with cancer of this region of the stomach, since both diseases are accompanied by gastric dilatation. The chief points of distinction between them are as follows: (1) In benign stenosis the symptoms of dilated stomach are almost invariably preceded by severe pain after food and by one or more attacks of hæmatemesis, while in cancer they develop after a few months of acidity, flatulence, and loss of flesh. (2) Hæmatemesis may occur in both diseases, but when it ensues from an ulcer it is occasional and copious, while in malignant disease the rejection of small quantities of altered blood is a frequent and characteristic phenomenon. (3) In the former complaint the loss of flesh, strength, and appetite is gradual, and the patient seldom displays the cachexia which accompanies a malignant growth of the stomach. (4) Simple ulcer is very rarely accompanied by a tumour; but the presence of an oval or round mass in the region of the pylorus is almost invariable in cases of cancer. (5) Benign stenosis is usually associated with hypersecretion of the gastric juice, and in the majority of cases an excess of free hydrochloric acid can be detected in the contents of the stomach. In cancer, on the other hand, the mineral acid is diminished or absent, while lactic acid is present in considerable quantity.

Adhesion of the pylorus to the gall-bladder also gives rise to dilatation of the stomach. In these cases, however, there is almost always a history of biliary colic or jaundice, and the

general symptoms of ulcer and cancer are absent.

Prognosis.—Stenosis of the pylorus is always a serious matter, and, according to the statistics of Gerhardt, is accompanied by a mortality of 10 per cent. As a rule death ensues from gradual exhaustion, but occasionally it occurs quite suddenly from syncope, or during a seizure of a tetanic or epileptic character. In other instances the failure of nutrition favours the inception of tuberculosis or some other intercurrent disease.

Treatment.—Whenever vomiting is a frequent symptom, rest in the recumbent position must be enforced. In less severe cases the patient should be directed to wear an elastic belt

Table 23.—The Differential Diagnosis of Cicatricial Stenosis of the Pylorus.

	Contents of stomach	Usually excess of free HCl, little or no lactic acid	Frec HCl, diminished or normal in amount	Free HCl absent, ex- cess of lactic acid	
	Physical signs	Stomach dilated; pylorus painful on pressure; tumour very rare	Stomach dilated; no tumour; tender-ness over pylorus, which is adherent to liver	Stomach dilated; tumourat pylorus; oval or round, smooth and tender; movable or fixed	
	Ameniu Loss of flesh	Gradual	Gradual	Rapid and severe	
,	Amenia	Moderate	Often ab- Gradual sent	Severe	
	Hæmatemesis	Occasional and profuse if ulcer un-healed	Absent	Frequent rejection of altered blood (coffeegrounds)	
	Pain	Moderate or ab- Occasional and sent profuse if ulcer un-	Constant; in right hypo- chondrium	Severe and lancinating; in abdomen and chest	
	History	For years pain after food, vomiting, or humatemesis, with tenderness in epigastrium. Corrosive poisoning	Formerly spasmodic attacks of pain; sometimes jaun- dice	For a few months wasting, pain, and indigestion	
	Disease	1. Cicatricial stenosis	2. External adhesions	3. Cancer of pylorus	

to support the dilated stomach, and he should avoid fatigue. In every instance excess of fluids with the meals must be prohibited, and only those foods be allowed which are easily digested and absorbed. Milk puddings, peptonised gruel, meat essences and jellies, the Leube-Rosenthal meat solution, scraped and pounded raw or lightly cooked meats, chicken, fish, poached or scrambled eggs, tripe, and sweetbread may be given in small quantities at frequent intervals. If retching and vomiting are incessant, recourse must be had to rectal alimentation.

The first indication for medicinal treatment is to remove the stagnant contents of the stomach and to cleanse its surface. With this object the organ should be washed out each morning before breakfast with warm water containing a drachm of bicarbonate of sodium to the pint. Care must be taken not to over-distend the viscus, and to thoroughly empty it at the conclusion of the operation. Various antiseptic solutions have been recommended for the purposes of lavage, but their employment is seldom necessary and is by no means devoid of danger.

At the commencement of the treatment the lavage should be performed each day, but after the first fortnight it may be necessary only two or three times a week. The patient should be taught to manipulate the tube for himself.

As soon as the stomach has been thoroughly cleansed and the diet suitably adjusted, an attempt may be made to control the processes of fermentation by the internal administration of antiseptic remedies. For this purpose carbolic acid, either in the form of the glycerine preparation (10 mins.) or as pure phenol (1 to 2 grs.), is invaluable when given two hours after meals. Some practitioners prefer to employ resorcine (10 to 15 grs.), the solution of perchloride of mercury (1 drachm), salicylic acid or salicylate of bismuth (10 to 20 grs.), creosote, B-naphthol, or salol, but none of them is so reliable as carbolic acid. When acidity is a troublesome symptom, bicarbonate of sodium, liquor potassæ, or calcined magnesia may be advantageously combined with the antiseptic remedy, while in those cases where the patient suffers from offensive eructations, powdered charcoal or charcoal biscuits are of considerable service. In every case the bowels require to be regulated by means of Carlsbad salts, cascara, or some other efficient aperient.

Surgical Treatment.—If the pyloric stenosis is moderate in degree, and the case comes under observation before the dilatation of the stomach is very pronounced, medicinal treatment is usually sufficient to arrest the progress of the complaint and to permit the patient to pursue his ordinary occupation; but should lavage combined with careful dieting fail to prevent vomiting, or the patient continue to lose flesh and strength, the question of surgical interference must be considered.

Of the several operations which have been devised to remove the obstruction, resection of the pylorus, partial or complete, has been practically abandoned. Loreta's digital divulsion has also been given up on account of its excessive mortality (38 per cent.), and the tendency of the stenosis to reappear. Gastroplicatio is also valueless, as it does not remove the cause of the dilatation. Pyloroplasty is a favourite operation with many surgeons, and has been followed by a mortality varying from 7 to 15 per cent. At the present day, however, gastro-enterostomy appears to be preferred by most operators on account of the comparative ease with which it is performed, and the slight mortality (3.8 per cent., Carle) by which it is accompanied. It is also probable that the diversion of the gastric contents through the adventitious channel favours the healing of the ulcer, and allays the reflex spasm of the pylorus which is responsible for many of the symptoms of stenosis. (For a full account of these various operations the reader is referred to treatises which deal with the surgery of the abdomen.)

(2) Stenosis of the Cardiac Orifice.—Ulceration of the cardiac end of the stomach is usually of an acute character, which produces little or no displacement of the surrounding tissues as the result of its cicatrisation. In those cases, however, where a chronic ulcer develops in the immediate neighbourhood of the coophageal opening, the irritation of the food is apt to excite a spasmodic contraction of the sphincter, while the healing of the sore itself may almost obliterate the orifice.

That stenosis of the osophagus at or near its entrance into the stomach may arise from simple ulcer is well shown by the following cases:—

Case XXXIX. A sailor, 30 years of age, was admitted into the London Hospital for incessant voniting. It appeared from his history

that about two years previously he had begun to experience severe pain at the chest immediately after meals. Within a few months each mouthful of food seemed to stick in the gullet, and eventually he was obliged to vomit in order to obtain relief. These symptoms had gradually increased in severity, so that for the last six months he had been obliged to confine himself to liquids. Recently even milk and beef-tea had given rise to vomiting. He had always been a total abstainer, and had never suffered from syphilis.

On examination the patient was found to be extremely emaciated and feeble. The abdomen was retracted, and a round, hard, and somewhat tender mass was felt beneath the left costal arch. The



Fig. 50.—Chronic ulcer at the cardiac orifice attended by dysphagia. Death from hæmorrhage. (London Hospital Museum.)

outlines of the stomach could not be determined. The tongue was foul, the bowels confined, and the temperature subnormal. No disease was detected in the other organs of the body. When solid food was administered, the patient immediately complained of pain behind the lower end of the sternum, and within a few minutes the bolus was vomited. Milk and other forms of liquid nourishment, when given in teaspoonful doses, merely produced a sense of oppression at the chest, but if drunk in larger quantities were at once rejected. The passage of a soft tube excited severe pain in the left side of the chest, and its point became arrested at 18 inches from the teeth. When milk was injected through the tube it regurgi-

tated. By means of rectal feeding the patient's life was sustained for nearly a fortnight, at the end of which time he sank from exhaustion.

Autopsy.— The stomach was contracted beneath the left ribs and resembled a cricket ball in size and shape. At the cardiac orifice was a chronic ulcer of crescentic shape which involved nearly two-thirds of the sphincter and extended slightly into the œsophagus. There was no obvious stenosis. No signs of cancer were detected with the microscope, either in the ulcer or in the surrounding tissues.

In the above case the symptoms pointed to a stricture of the esophagus, and this conclusion was apparently confirmed by the arrest of the bougie in the vicinity of the cardiac orifice and by the discovery of a tumour beneath the left ribs. It was shown, however, after death that the curious contraction of the stomach was the cause both of the tumour and the apparent obstruction, so that it can only be supposed that the dysphagia was due to spasm of the muscular structure at the seat of disease.

Case XL. A female, 42 years of age, was admitted into the London Hospital for pain and vomiting. Her history indicated that she had enjoyed good health until about a year before, when she noticed that solid food often gave rise to pain at the lower part of the chest. This symptom gradually increased until every mouthful of food she swallowed appeared to lodge in the gullet and produced a painful spasm which threatened to choke her. For several months she had lived on liquids, but latterly the act of swallowing had become more difficult and the greater part of each meal had been vomited at once.

On admission the patient was extremely feeble and profoundly emaciated. The abdomen was retracted, and the stomach appeared to be small in size. No tumour to be felt. A bougie inserted into the esophagus encountered an obstruction just above the stomach. All forms of nourishment were immediately rejected, and death ensued in the course of a few days.

Autopsy.—The upper border of the stomach was adherent to the left lobe of the liver. At the cardiac orifice was a chronic ulcer about the size of a florin, the contraction of which had twisted and displaced the lower end of the æsophagus. The peritoneal surface of the ulcer was considerably thickened, and the contiguous margins of the stomach and æsophagus were firmly united by fibrous tissue. The distortion and compression produced in this manner had practically obliterated the lumen of the last half inch of the tube.

In the next case the ulcer was probably situated in the cesophagus, but the absence of an autopsy rendered it im-

possible to determine the exact site of the disease. The early age of the patient and the character of his symptoms render it extremely improbable that the stricture was malignant in character.

Case XLI. A clerk, 23 years of age, was admitted into the London Hospital for pain and vomiting. About twelve months previously he had commenced to suffer from acute pain behind the lower end of the sternum and in the left side of the chest after swallowing meat, and this symptom had gradually increased in severity, so that for the last six months he had been obliged to restrict himself to liquid nourishment. Soups and beef-tea containing salt had also caused pain, and on several occasions the act of deglutition had been followed by a choking sensation and regurgitation of the food. Latterly the dysphagia and vomiting had become more frequent. He had lost nearly three stones in weight within two months. He had never been a hard drinker, nor had he suffered from syphilis. There was no history of hæmatemesis.

On examination the patient was found to be extremely emaciated, but not markedly anæmic. The tongue was foul, the pulse weak, and the temperature subnormal. There was no evidence of disease of the lungs or of thoracic aneurism. The abdomen was retracted, and the stomach small in size, but neither tumour nor localised tenderness could be detected. The urine was diminished in quantity, but was devoid of sugar and albumen. When a soft tube was passed into the œsophagus its point encountered a slight sense of resistance at a distance of $16\frac{3}{4}$ inches from the teeth, and at the same time the patient complained of intense pain behind the lower end of the sternum and in the left side of the chest. With the aid of a little manipulation, the obstruction was overcome and the tube passed into the stomach, but the pain continued for several minutes after the instrument had been withdrawn. Every attempt to swallow solid food was followed immediately by a similar attack of pain, and within a few seconds by retching and vomiting. The same symptoms ensued after the ingestion of hot milk, but when cold fluids were sipped slowly the deglutition sound could be heard over the epigastrium within twenty seconds. After two ounces of milk had been swallowed in this manner a soft tube was inserted as far as the tender spot, and about six drachms of uncurdled milk mixed with mucus and saliva were withdrawn from the œsophagus. On one occasion a little bright blood was evacuated, but no evidence of a morbid growth was detected on microscopic examination of the material.

After rectal feeding had been employed for a few days the stomach tube was coated with cacao butter containing 5 per cent. of

hydrochloride of cocaine, and peptonised foods were injected into the stomach every six hours. The patient soon learnt to feed himself, and gained twenty-one pounds in weight in four weeks, at the end of which time he returned to his home in the country, having declined to undergo the operation of gastrostomy. We subsequently ascertained that after a few months he relinquished the use of the tube, and eventually died from inanition.

From these and other similar cases which have come under our notice we are inclined to believe that simple ulceration near the cardiac orifice is not infrequently the cause of dysphagia and other symptoms indicative of stenosis of the lower end of the œsophagus. The complaint is rather more common in men than in women, and is chiefly encountered between the ages of twenty-five and forty. It is sometimes associated with cirrhosis of the liver.

The earliest symptom to attract attention is pain at the chest after food. At first it is only experienced after the ingestion of solid food, and is chiefly referred to the lower end of the sternum or to a corresponding spot in the back, but subsequently it may radiate round the left side of the chest and continue for many minutes after the bolus has reached the stomach or been rejected. Gradually the symptom becomes more frequent, so that after the lapse of a few months the patient is afraid to indulge in ordinary food, and restricts himself to milk and other forms of liquid nourishment. Hot fluids and those containing salt, pepper, or other condiments may also give rise to pain.

Finally this symptom is accompanied or replaced by a feeling of obstruction in the gullet, and every attempt to swallow is followed by choking or retching, and finally by vomiting. In advanced cases the dysphagia may be almost complete, and the food regurgitates as soon as it is swallowed. The inability to take sufficient nourishment occasions rapid loss of flesh and strength, and soon the patient presents the appearance of starvation. The bowels are confined, the pulse is feeble, and the temperature falls to a point considerably below the normal. Occasionally hæmatemesis or melæna occurs. Cachexia, like that which accompanies malignant disease of the æsophagus, is never observed.

Physical Signs.—The abdomen is markedly retracted, and it is often possible to feel the anterior surfaces of the vertebrae

and to trace the abdominal agrta throughout its course. The stomach is smaller than normal, and it may be impossible to determine the outlines of the viscus. In case 39 the contracted stomach gave rise to a palpable tumour, but this condition is extremely rare. When the patient swallows a mouthful of liquid the deglutition sound is much delayed, or may even be suppressed altogether. Exploration of the esophagus with a soft tube often gives rise to pain at a spot about sixteen inches (forty centimetres) from the teeth, which may continue for some time after the instrument has been withdrawn. In the early stages of the complaint some obstruction can usually be felt, but this is easily overcome by a little pressure (spasm). At a later period the signs of stenosis become more pronounced (cicatrisation). Owing to the existence of dilatation of the esophagus above the seat of stricture, it is often possible to evacuate several drachms of alkaline food mixed with saliva and even blood. Microscopic examination proves that the material is free from particles of cancerous tissue. The contents of the stomach often contain an excess of hydrochloric acid.

Prognosis.—Stenosis of the cardia always terminates fatally unless some means can be devised to dilate the stricture or to preserve the nutrition of the patient. The duration of the disease when left to itself varies from a few months to several years, according to the degree of obstruction. It usually pursues a rapid course when it arises from ulcer of the esophagus, or

from corrosive poisoning.

Diagnosis.—The disease has to be distinguished from two other affections of the lower end of the esophagus, namely, cancer and spasmodic stricture. Stenosis from ulcer is most frequent between twenty-five and forty years of age, and is invariably preceded by symptoms of ulcer of the stomach, such as severe pain after food, vomiting, or hæmatemesis: cancer, on the other hand, is usually encountered after the age of forty, and is principally characterised by progressive dysphagia. In the former disease the contraction of the orifice occurs very gradually, and two or more years may elapse between the initial symptoms of the complaint and fatal inanition. Cancer develops much more rapidly, and loss of flesh and cachexia are early phenomena. It should also be observed, that the material evacuated from the resophagus

Table 24.—The Differential Diagnosis of Stenosis of the Cardiac Orifice

Contents of association	Saliva, mucus, unaltered food; no fætor	Fetid; often gru-mous; some times particles of cancerous tissue	Nil, or a little mucus and saliva	
Exploration Contents of asophagus	Painful if ulcer open; stricture seldom complete, often spassmodic	Stenosis steadily increases till only fine bou- gies pass	Often gives rise to hysterical symptoms; large bougies pass more easily than small	
ition ds	Delayed	Delayed or absent	Vary from day to day	
Hemor-	Profuse in early stages	Occasionally in small quantities	Absent	
Anemia	Often absent	Cachexia	Often chlorosis	
Loss of flesh	Gra- dual	Rapid and pro- gres- sive	Varies	
Vomiting (recurgitation) of food	Chiefly after solid food	At first after solids; subsequently after liquids	Irregular or absent	
Pain	Severe after food if ulcerun- healed	Dis- comfort after food	Absent	
us Onset and Pain (regurgita- Loss of Anæmia rhage soun food	Onset gradual; lasts 6 months to many years	Onset rapid; lasts 9 to 12 months	Onset often sudden; symp- toms inter- mittent	
Previous listory	Symptoms of gastric ulcer	1	Hysteria, neuras- thenia, or excess of alco- hol	
Age	Usually before	After 40	18 to 30	
Sex		Males	Females	
Disease	Stenosis from ulcer	Stenosis from cancer	Simple spas- modic stricture	

above the stricture is often fetid in cancer and contains minute pieces of the morbid growth.

Simple spasmodic stricture of the cardiac orifice is chiefly met with in women from eighteen to thirty years of age, who are the subjects of anæmia, hysteria, or neurasthenia. The complaint may last for many years, but varies greatly in intensity from time to time. Hæmatemesis never occurs, and the vomited matters are not fetid in character. With a little care it is usually possible to pass a full-sized tube through the obstruction. The disease is readily amenable to treatment.

Treatment: Medical.—The difficulty of swallowing necessitates the employment of liquid nourishment, such as milk. soups, meat essences, meat solutions and jellies, with meat powder and peptone, which may be given in small quantities at short intervals. A large nutrient enema should also be administered every six hours. Whenever it is possible a soft tube should be introduced into the stomach once or twice a day, not only for the purpose of feeding, but also with the view of preventing the stricture from becoming impermeable. If the instrument gives rise to pain, it must be used with great caution lest it excite hæmorrhage or perforation. Smearing the lower end of the tube with cacao butter containing 5 per cent. of hydrochloride of cocaine is often of value under these conditions. If the obstruction appears to be chiefly dependent upon muscular spasm, a mixture containing bromide and iodide of potassium with tincture of belladonna may be prescribed, or an injection of morphine and atropine may be given. Occasionally a lozenge containing cocaine and morphine swallowed just before meals is of value.

In every case the mouth must be carefully cleansed at intervals with a mild antiseptic wash, and the bowels maintained in regular action by enemata or small doses of aperients.

Surgical.—If the symptoms of ulceration have subsided, an attempt may be made to dilate the stricture by means of bougies, but this must be done in a very gradual manner and without the use of undue force. When the instrument has entered the stomach it should be allowed to remain in position for an hour or two if possible.

Whenever the stricture is impermeable, or severe pain prevents the ingestion of food, gastrostomy should be performed at once. As a rule this operation is postponed until the patient

THE RESULTS OF CICATRISATION CHIRUHCIGAL SUCIETY

is almost moribund, when its chances of success are extremely limited; but when it is undertaken at an early stage of the complaint it not only removes the symptoms of inauition, but affords rest to the diseased structures, and thereby allays the muscular spasm which plays such an important part in the production of the dysphagia. Out of the twenty-six successful cases of gastrostomy for cicatricial stenosis of the cardia recorded by Le Fort, thirteen eventually recovered their power of swallowing. If the stenosis is due to a fibrous contraction of the cardiac orifice, it may be possible to dilate the stricture after the patient has recovered from the effects of the operation. It is usually a difficult matter to determine the position of the esophageal opening through the abdominal wound, but in one case Loreta was able to practise digital divulsion with success. Socin has suggested that the patient should swallow a leaden shot attached to a firm thread, which can afterwards be removed through the fistula and serve as a guide for a fine bougie, and he has published one case in which this method was employed with advantage.

(3) Hour-glass Deformity of the Stomach.—This condition is a rare result of chronic ulceration, and is chiefly confined to women from thirty-five to sixty years of age. The majority of the recorded cases of 'hour-glass' stomach are examples of the congenital malformation with secondary ulceration in the vicinity of the stricture (Carrington, Sievers).

In every instance the patient has suffered from symptoms of gastric ulcer for a long period; in one of our cases severe pain after food, with vomiting and occasional attacks of hæmatemesis, had existed for six years, while in others which have been published the length of time that elapsed between the initial signs of ulcer and the death of the patient varied from five to twenty years.

The symptoms which accompany this deformity of the stomach are partly due to the ulcer, which often remains unhealed, and partly to chronic catarrh of the gastric mucous membrane; but when the constriction between the pyloric and cardiac portions of the viscus has become extreme, vomiting makes its appearance and continues until death.

Pain after food is almost invariable, and in the majority of cases exhibits the characteristic features of that which accompanies gastric ulcer. In others, distension of the abdomen is the chief cause of complaint, while occasionally the patient appears to be conscious of the peristaltic movements of her stomach (Case 42).

Vomiting is a somewhat later symptom, and at first occurs only at the climax of the painful crisis; but after a time it takes place every day, as in cases of pyloric stenosis. The ejecta usually contain an excess of hydrochloric acid. Exposure to cold, or an indiscretion of diet, is apt to induce an attack of subacute gastric catarrh, which gives rise to incessant retching and vomiting, and may occasion profound and even fatal exhaustion. Under these circumstances the vomited matters consist entirely of alkaline or bile-stained mucus.

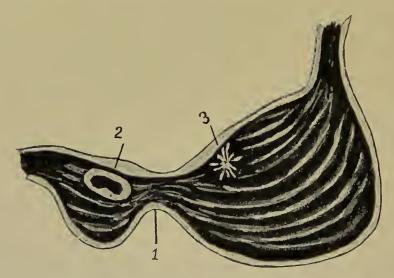


Fig. 51.—Sketch of an hour-glass stomach of congenital origin. 1, site of the constriction; 2, large chronic ulcer in the pyloric region; 3, scar of a former ulcer. (From a case at the London Temperance Hospital.)

Hæmatemesis and melæna are not infrequent, and sometimes result from secondary ulceration of the stomach near the stricture (fig. 51).

In the early stages of the complaint the appetite is usually unimpaired, and in one case it was observed that the patient consumed enormous quantities of food, but was forced to eat very slowly. At a later period, when vomiting is frequent, the desire for food diminishes, and may be replaced by complete anorexia. The condition of the tongue varies, being sometimes clean and red, while at other times it is thickly coated with a creamy fur. The breath is often offensive, and eructations of fetid gas and

acid fluids are a frequent source of annoyance. The bowels are confined, the urine diminished in quantity, and the temperature subnormal. An excessive secretion of saliva is occasionally observed. In every instance the patient becomes extremely emaciated and feeble, and death ensues either from chronic inanition or from exhaustion after vomiting. The principal features of the disease are well shown in the following cases.

Case XLII. A woman 53 years of age was admitted into the Cornwall Royal Infirmary in 1872 for disease of the stomach, under the care of Dr. Jago. For 29 years she had been a widow, and had ceased to menstruate at the age of 39. For 17 years she had acted as nurse in the Infirmary, the arduous duties of which office she had performed with undeviating diligence up to a few months before her death, and was invariably equal to her work. At the age of 30 she had suffered for about two years from severe vomiting, but was uncertain whether blood had been vomited or if there had been much pain after meals. Eight years before the present illness she was again attacked by troublesome vomiting, but there was no gastralgia or hæmatemesis. Her appetite was said to be excessive, but she required a long time to eat her meals. Ten months previously she became liable to occasional vomiting; this symptom increased in frequency until at the end of four months scarcely anything she swallowed was retained in the stomach more than half an hour; as the result of this she rapidly lost flesh and became very debilitated.

On examination the various thoracic viscera were found to be perfectly healthy. There was no pain or tenderness in the abdomen, and no tumour could be detected. The tongue was clean, the appetite unimpaired, and the only sensation complained of after meals was "rolling round of the food in the stomach" until it was painlessly ejected after the lapse of an hour or so. The food presented no signs of digestion and was not acid in taste.

During the last three months of her life every expedient was resorted to in order to relieve the vomiting, but without avail. About a week before death, however, the emesis ceased, but she lost all relish for food and became troubled with frothy saliva.

Autopsy.—The only morbid organ was the stomach, which was divided into two pouches and contained 3 ounces of a slightly acid chocolate-coloured fluid. When the viscus was laid open the stricture was found to admit the index finger. On its pyloric side was a large chronic ulcer and a scar, the contraction of which had presumably caused the deformity.

Case XLIII. A married woman, aged 46 years, was admitted to King's College Hospital on November 8th, 1897. The family history was unimportant. In December 1881 the patient began to

suffer great pain in the hypochondriac region and in the right shoulder, and, in fact, with all the symptoms of ulcer of the stomach, with the exception of hæmorrhage. She was treated for this condition by diet and in other ways, but had suffered from symptoms off and on. Sometimes she would be pretty well for a few months, and then without apparent cause the whole trouble would return again. Latterly the symptoms had been those of obstruction rather than ulceration, and the diagnosis of pyloric stricture had been made by several physicians. The patient was very considerably emaciated and was in a feeble state of health. She did not so much complain of pain in the hypochondriac region as of what she termed "an uncomfortable feeling"—a sense of distension, flatulence and frequent vomiting. The vomiting occurred every second or third day, but she did not bring up anything like the whole quantity of food she took. She had lately been living entirely upon liquids. The amount she would vomit after a couple of days varied from 16 oz. to 30 oz.; the longest time she had gone lately without vomiting was three days. Her weight on admission was 7 st. $4\frac{1}{2}$ lbs.

On examination the stomach was not very much dilated, and the dilatation was not uniform; it appeared to be specially marked about the cardiac end and to a less extent towards the pylorus. On listening with a stethoscope over the centre of the stomach a gurgling sound could be heard from time to time as if fluid was running through a narrow orifice, and Dr. Burney Yeo, who saw the patient in consultation, stated that in his opinion the case was one of hour-glass stomach resulting from an old ulcer. On December 1st, Mr. Watson Cheyne made a vertical incision over the stomach, in the middle line, about four inches in length, and the organ was drawn out of the wound and opened. It was then found to be considerably dilated in its cardiac portion, and to be divided about its centre by a stricture which would only admit the passage of a quill. There was a good deal of scarring and cicatricial tissue in the neighbourhood, and at the actual point of constriction there was no mucous membrane at all.

An incision was made through this contracted portion in the line of the stomach, so that the ring of cicatricial tissue was divided transversely; this incision was extended into the healthy parts of the stomach for about one and a half inches on each side, and then the angles of the incision were brought together and the rest of the part was sewn up; in fact the operation was identical with that of pyloroplasty. After the operation an opening was left between the two portions of the stomach which could readily admit a couple of fingers.

At the end of a fortnight she commenced to take solid food, and was discharged on January 7th, having gained 8 lbs. in weight. This

improvement still continues, and at the present time (March 1898), although somewhat troubled with flatulence, her condition is practically normal. (Recorded by Mr. Watson Cheyne.)

Diagnosis.—It is rare for the deformity of the stomach to be recognised during life. In a case reported by Eichorst, the peculiar conformation of the organ was visible through the attenuated abdominal wall, and it is possible that inflation of the stomach with air or gas might render this mode of

diagnosis of considerable value.

In most cases the epigastrium is tender on pressure, and the stomach appears somewhat dilated. It may usually be observed, however, that the enlargement chiefly affects the cardiac portion of the organ, and this fact, combined with the detection of peristaltic movements confined to the left hypochondrium, greatly aided the diagnosis in our case. In one instance, Jaworski states that a splashing sound could be obtained by palpation after the stomach had been apparently evacuated by the siphon, owing to the retention of fluid in the pyloric pouch ('paradoxical dilatation.') Lavage is also productive of another sign of some significance. After the organ has been thoroughly cleansed a gush of undigested food may take place through the stricture, and render the fluid suddenly foul. This sign, however, is not peculiar to the complaint, as it is also met with in cases of gastric diverticula or where an abscess of some neighbouring viscus communicates with the cavity of the stomach.

In Case 43 a gurgling sound was heard over the centre of the stomach, as though fluid was being forced through a narrow channel.

Treatment.—In the early stages the symptoms may usually be controlled by careful dieting, lavage, and the employment of sedatives and antiseptics; but when the patient's condition is endangered by constant vomiting, surgical interference becomes necessary. In Case 43 the stricture was divided and the wound sutured, as in the operation of pyloroplasty, but some surgeons prefer to establish a gastric anastomosis, or to perform gastro-enterostomy. The results of operative interference have been highly successful (Hochenegg, Schmidt-Monard).

Obliteration of the Bile Duct.—A chronic ulcer situated over the biliary papilla in the duodenum frequently gives rise

to persistent jaundice by obstructing the outflow of the bile. In most instances the obstruction is incomplete, and after death a probe can be passed into the duct, or bile can be expressed from it. This probably explains the variations in the colour of the skin mentioned in some of the recorded cases, as well as the occasional disappearance of the tumour formed by the enlarged gall-bladder. When the obstruction is due to cicatrisation of the ulcer, the jaundice is severe and permanent. As a rule the icterus appears somewhat suddenly, and is accompanied by slight enlargement of the liver and by gradual emaciation. Death usually ensues from exhaustion within three years, but it may occur from coma at a much earlier period. Beyond a history of dyspepsia there is seldom any indication of the existence of a duodenal ulcer.

Case XLIV. A man 48 years of age, was admitted into the London Hospital for jaundice and emaciation. He stated that he had been in good health until about four months before, when he noticed that his skin was turning yellow and he suffered from flatulence after food and constipation. He had never had pain after meals, vomiting or hæmatemesis, nor was there any history of biliary colic. On examination the liver was found to project rather more than an inch below the costal margin, and presented a sharp edge and a smooth surface. There was no pain on deep palpation over the region of the gall-bladder. The spleen was normal in size and there was no ascites. The temperature was subnormal. The patient grew gradually weaker, and the jaundice became more intense. appetite failed, emaciation proceeded rapidly, and he died comatose about eight months after the onset of his illness. Occasionally a sense of tumour was felt in the region of the gall-bladder, but it was never definite.

Autopsy.—The liver was somewhat enlarged and deeply bile-stained. The gall-bladder was distended and its walls thickened. The common duct was enormously dilated, and all the hepatic ducts were much enlarged. In the duodenum, situated directly over the biliary papilla, was a large chronic ulcer which had become partially cicatrised. The opening of the bile-duct was not completely obstructed, as bile could be squeezed through it. The pancreatic ducts were immensely dilated.

Case XLV. A woman, aged 58, was admitted into Guy's Hospital under the care of Dr. Pavy, for jaundice and enlarged liver. She died fifteen months after the onset of the jaundice, at which time her skin was of a dark greenish hue, and she was extremely emaciated.

Autopsy.—The liver was only slightly enlarged. The ducts were all immensely dilated and filled with dark green bile. The hepatic duct projected outwards in a spiral manner, and was as large as the intestine of a child. The gall-bladder was also greatly distended, reaching far below the liver and forming the tumour felt during life. The common bile duct was four inches long and much dilated. The dilatation continued as far as the duodenum, where it suddenly became contracted within an eighth of an inch from the interior of the bowel. As a probe could be passed into it from the bowel, and bile could be squeezed out, it was not perfectly occluded. The end of the duct felt thickened, and the contraction was evident when the tube was laid open. It appeared like the contraction that would ensue from a minute ulcer. The mucous membrane was irregular on the surface, and the submucous tissue thickened. The pancreatic duct was healthy. [Perry and Shaw.]

Case XLVI. A man, aged 68, had always enjoyed excellent health until he was suddenly seized with jaundice, and subsequently had repeated attacks of fever. The jaundice recurred several times, and three years later he came under the care of Dr. Stokes. During the last eight days of his life the fever continued without intermission, and he died comatose.

Autopsy.—The bile ducts were enlarged, and presented aneurysmal dilatations in the liver; the common bile duct was also dilated, and its orifice in the duodenum was surrounded by an irregular fungus resembling an old cicatrix. The obstruction was not complete, for the orifice, although constricted, was not obliterated.

Case XLVII. A woman, aged 68, was admitted into St. Thomas's Hospital with jaundice and emaciation of seven months' duration. The gall-bladder could be felt occasionally. She died four months after admission.

Autopsy.—The gall-bladder was much enlarged and its walls thickened. The common bile duct was greatly dilated and its orifice was obstructed by a partially healed ulcer in the duodenum, about the size of a sixpence. The pancreatic duct was also much dilated.²

Treatment.—Whenever medicinal treatment fails to relieve the jaundice, or when from the absence of physical signs the cause of the complaint is obscure, an exploratory incision should be made with the view of exposing the gall-bladder and common bile duct.

Dublin Journal, N.S., 2, p. 505. Cited by Perry and Shaw.

² St. Thomas's Hospital Reports, N.S., vol. 13, p. 435. Cited by Perry and Shaw.

LITERATURE ON HOUR-GLASS STOMACH.

Bauermeister, Inaug. Dissert., Halle, 1890. Carrington, Path. Soc. Trans., vol. xxxiii. p. 130. Cheyne, Lancet, 1898, vol. i. p. 785. Chiari, Wiener med. Wochenschrift, 1890, No. 42. Hacker, Wiener mcd. Wochenschrift, 1883, No. 37. Hirsch, Virch. Archiv, 140, 3, p. 177. Hudson, Path. Soc. Trans., vol. xxxviii. p. 133. Jago, Medical Times and Gazette, 1872, vol. ii. p. 409. Jaworski, Wiener klin. Wochenschrift, 1888. Kern, Inaug. Dissert., Berlin, 1891. Mazotti, Rivista Clinica di Bologna, Aug. 1824. Palmer, Medico-Chirurgical Journal, Jan. 1816. Reiche, Jahrb. d. hamburg. Staatskrankenanstalt, 1890, p. 180. Rémond, Gaz. des Hôpitaux, Nov. 14, 1891. Roger Williams, Journ. of Anat. and Physiol., vol. xvii. p. 460. Saundby, Deut. med. Wochenschrift, 1891, p. 382. Sievers, Berl. klin. Wochenschrift, April 10, 1899. Stoker, Med. Press & Circular, March 3, 1869. Struthers, Anatomical and Physiological Observations, Edinburgh, 1854.

Tilger, Virchow's Archiv, Bd. 123, Heft 2.

CHAPTER III

HYPERSECRETION AND GASTRIC CATARRH

EVERY practitioner is aware that persons who have suffered from ulceration of the stomach are liable to various functional disorders of digestion for a long time after the original complaint has been cured. The acute form of the disease in young women is usually followed by the symptoms of gastric atony with deficient secretion of hydrochloric acid, but after the cicatrisation of a chronic ulcer, hypersecretion (gastrosuccorrhoea) and gastric catarrh are very apt to occur, and may prove more troublesome than the primary disease.

It will be noticed that a distinction is here made between catarrh of the stomach and hypersecretion, notwithstanding the fact that it is the custom to ascribe the continuous secretion of gastric juice to the hypertrophy and proliferation of the glands, which have been described by the terms 'catarrhus acidus' (Korczinski and Jaworski), and 'gastrite hyperpeptique' (Hayem).

In discussing the etiology of gastric ulcer we ventured to state our dissent from the view that chronic hypersecretion was the principal factor in the production of the disease, and expressed the opinion that the abnormal functional activity of the stomach is merely a reflex expression of chronic irritation of its mucous membrane. We would now go a step further and state our emphatic belief that the so-called disease of Reichmann, or gastrosuccorrhoea, has no claim to be regarded as a morbid entity, but is merely a symptom of chronic irritation of the gastric nerves, either by an ulcer or by food which has been retained in the viscus. Our reasons for this unorthodox opinion are shortly as follows: (1) The chief diagnostic sign of hypersecretion is the discovery of gastric juice in the fasting stomach, it being believed that under ordinary circumstances the viscus is empty in the early morning. Schreiber, however, has recently shown that in

nearly 70 per cent. of his patients in Königsberg, gastric juice containing hydrochloric acid and ferments could be extracted from the organ before breakfast, and in every case which we have investigated a similar result was obtained, whether the stomach had been washed out on the previous evening or not. This continuous secretion is probably due to the fact that most people swallow saliva and mucus during the night, the alkalinity of which serves to stimulate the gastric glands, for it is particularly noticeable in those cases of early phthisis where the patient is in the habit of swallowing his expectoration. (2) The phenomenon is invariable during the early stages of dilatation of the stomach, whether the lesion be due to atony of the gastric walls, to stenosis or spasm of the pylorus, or to obstruction of the duodenum; but it usually disappears as soon as chronic catarrh of the mucous membrane has become established. This latter fact explains the usual absence of free hydrochloric acid in cancer of the pylorus, and in cases of cicatricial stenosis from corrosive poisoning. It is probable that the retention of even a little food in the dilated viscus is sufficient to induce a more or less continuous secretion, since, after the most careful lavage overnight, particles of food can usually be extracted in the early morning. (3) Hypersecretion accompanies the majority of chronic ulcers of the stomach, but it usually disappears when the disease heals. When, however, the process of cicatrisation produces stenosis of the pyloric orifice with consecutive dilatation of the organ, the disorder not only continues, but usually becomes intensified. This is the result partly of retention of food and partly of irritation of the gastric nerves from their implication in the cicatrix. (4) In every case of 'Reichmann's disease' which we have examined after death, there has been either stenosis of the pylorus or duodenum, the existence of which was unsuspected during life, or an hypertrophied state of the gastric walls which suggested the previous existence of a functional obstruction (spasm). (5) Carle and Fantino have shown that whenever the symptoms of hypersecretion are associated with stenosis of the pylorus they disappear as soon as the obstruction has been removed by operation or the retention of food prevented by gastro-enterostomy, while, on the other hand, idiopathic cases of Reichmann's disease may usually be cured by a similar operation.

We will now proceed to discuss the various symptoms of

the hypersecretion and gastric catarrh which usually follow the

cicatrisation of a chronic ulcer near the pylorus.

Pain in the abdomen is complained of in every case. This symptom differs from that of ulcer by occurring two or three hours after meals, and being temporarily relieved by milk, eggs, or other kinds of nitrogenous food. During the early stages of the complaint the patient complains of an aching or burning sensation in the epigastrium, which subsides spontaneously after an hour or two; but subsequently the pain grows more violent, and radiates over the greater part of the abdomen and chest. The attacks are especially prone to occur between the hours of 11 P.M. and 4 A.M., while sometimes the pain is either continuous or temporarily relieved after each meal. Towards the termination of an attack flatulent distension of the abdomen with pyrosis and substernal cramp are frequently present.

Vomiting is an almost invariable symptom of the complaint. At first it may occur only during the night, when it serves to relieve the pain by ridding the stomach of a large quantity of acid fluid mixed with undigested food, but after a time it becomes more frequent, and in advanced cases may take place two or three times a day. Occasionally the vomiting is replaced by a sudden attack of diarrhoa, after which the pain and pyrosis rapidly subside. The ejecta vary in quantity from 10 to 40 ounces, and consist of a yellow or brown fluid mixed with undigested food. When vomiting occurs in the early morning the material has often a green tinge owing to the presence of altered bile, and in long-standing cases it is largely mixed with mucus. After filtration a clear sour-smelling fluid is obtained which gives a brilliant reaction with the phloroglucin-vanillin test. The total acidity varies from 40 to 90, and the hydrochloric acid from 0.1 to 0.4 per cent. During the later stages of the complaint the acidity gradually diminishes, and the free mineral acid may completely disappear. Lactic acid seldom exists except when chronic catarrh is a feature of the case. Peptone and erythrodextrine are easily demonstrated in the filtrate, and if a sufficiency of hydrochloric acid is present the fluid exerts a digestive action upon eggalbumen. Microscopic examination of the residue upon the filter paper reveals the presence of starch granules, globules of fat, small pieces of muscle-fibre, bacilli, sarcing, epithelium, and not infrequently peculiar spiral bodies formed of mucus.

The bowels are confined at first, and the stools are pale and hard, and passed with difficulty. This tendency to constipation is probably due, partly to neutralisation of the bile and the other secretions in the intestine by the abnormally acid chyme, and partly to the vomiting which reduces the quantity of fluid in the circulation. At a late stage of the complaint attacks of diarrhœa are apt to supervene from secondary catarrh of the intestine, and the evacuations are then mixed with an excess of mucus, and occasionally with blood.

The *urine* presents several features of interest. Owing to the frequent vomiting the quantity is greatly diminished, and seldom exceeds 20 ounces in the course of the twenty-four hours, while in severe cases only 4 or 5 ounces may be voided in the same period of time. It is usually pale yellow in colour, slightly alkaline in reaction, and often presents a turbid appearance from the presence of phosphates. The

specific gravity varies from 1020 to 1030.

As a result of the increased functional activity of the stomach the daily excretion of urea is increased both relatively and absolutely, as much as 950 grains being often voided in the course of twenty-four hours. Phosphates are also eliminated in greater quantity than under normal conditions. Crystals of uric acid and amorphous urates are usually present in the deposit, and peptones can often be recognised by appropriate tests. One of the most noticeable features of the urine is the marked diminution in the amount of chlorides it contains. Under normal conditions these constituents of the urine slightly diminish during the period of gastric digestion, owing to the requisition of the sodic chloride in the blood for the manufacture of hydrochloric acid; but in cases of hypersecretion, where the stomach is constantly manufacturing a secretion rich in chlorine compounds, which is subsequently lost by vomiting, the chlorides may almost entirely disappear from the urine.

The tongue seldom exhibits any notable deviation from the normal in the early stages of the complaint, but when vomiting occurs frequently it becomes coated with a creamy fur, and the breath acquires a sour and offensive smell. This latter condition is sometimes associated with a rapid decay of the teeth, which appears to be due to fermentation of the unhealthy buccal secretions.

As long as the disease is uncomplicated with gastric catarrh the appetite is preserved, and is often increased. Occasionally the patient complains of extreme hunger during the night, and will leave his bed in search of food, despite the knowledge that such indulgence will inevitably lead to severe pain. This intense craving is only relieved by nitrogenous food, and, as a rule, little desire is expressed for articles of a saccharine or starchy character. In advanced cases the appetite is completely lost, and the anorexia may be as complete as in cancer of the stomach.

Thirst is an invariable symptom of the complaint, and is often excessive. It is usually most pronounced at night-time, or when vomiting is frequent. Large draughts of cold water are most agreeable to the patient, but the relief they afford is only temporary.

Anæmia is always present, and gradually increases as the disease progresses. Examination of the blood shows that there is a marked diminution in the amount of hæmoglobin, but the percentage of red corpuscles is seldom much reduced.

In all cases the pain and vomiting after meals produce a condition of general malnutrition, so that the patient loses flesh and strength. In the later stages of the complaint emaciation is sometimes so extreme as to rival that which attends cancer of the stomach. At this period dyspnæa occurs upon exertion, and ædema of the ankles is often observed. The pulse is feeble and unduly slow, and the heart sounds are indistinct. Hæmic murmurs are sometimes audible over the pulmonary and mitral areas. The temperature of the body is subnormal. As the prostration increases the patient becomes fretful, impatient, and emotional, and occasionally suffers from delusions. Insomnia is a frequent and troublesome symptom; and complaint is sometimes made of twitching of the muscles of the extremities or face, and of a feeling of formication in the limbs.

The constant presence of the hyperacid secretion tends to irritate the mucous membrane and excites chronic gastric catarrh. The symptoms of the latter complaint are masked by those of the functional disorder, so that it usually escapes notice until the supervention of an acute attack.

When this occurs the patient suffers from constant nausea with incessant retching and vomiting, which prevent the

administration of any nourishment by the mouth. The vomit rapidly loses its usual characters, and consists of a glairy alkaline fluid mixed with saliva and bile, or of tenacious mucus. The appetite is completely lost and the patient may even cease to complain of thirst. The tongue is thickly coated with a white fur, the pulse is slow and feeble, and the temperature of the body depressed. Throbbing in the head, faintness and palpitation frequently follow an attack of retching. In most instances the bowels are confined, but occasionally diarrhoea is observed from catarrh of the intestine. The urine is diminished in quantity and contains an excess of phosphates. Jaundice from catarrh of the bile duct and laryngeal catarrh are occasional sequelæ of an attack. In every case the inability to retain food gives rise to great exhaustion and rapid loss of flesh, and in debilitated subjects death frequently occurs from failure of the heart. The duration of an acute attack varies from three to fourteen days or even longer, and the complaint exhibits a tendency to recur every few weeks. As a rule no cause can be assigned for its sudden recrudescence, but occasionally it appears to be excited by certain articles of diet, and is particularly prevalent during the spring and autumn months of the year. The general features of the complaint are well shown in the following case.

Case XLVIII. A gentleman, about 35 years of age, began to suffer from pain after solid food and vomiting which gradually increased in severity until nine months later he was attacked by violent hæmatemesis. After confinement to bed and strict dieting the symptoms of ulceration subsided, so that at the end of about eighteen months from the commencement of the disease he was considered to be eured. As soon, however, as he resumed his former mode of life and no longer restricted himself with regard to his diet, he experieneed flatulence and acidity during the night which seriously interfered with sleep. These symptoms steadily increased until he was obliged to induce vomiting in order to obtain relief. Subsequently a burning pain at the epigastrium, followed by acidity, flatulenee, and nausea, and occasionally by vomiting, ensued regularly twice a day, about four o'clock in the afternoon and eleven at night. The appetite was well preserved, and after vomiting had occurred a feeling of intense hunger was often experienced. Dryness of the mouth and thirst were a constant source of discomfort, and the bowels were very confined. He observed that the pain and acidity eould be temporarily relieved by draughts of cold water or milk, and were often removed by large doses of biearbonate of sodium. After these symptoms had lasted

for nearly a year he was suddenly attacked by nausea and vomiting, which continued for nearly a week and prevented him from taking any food. When the sickness subsided he remained very prostrate and was found to have lost eight pounds in weight. By restricting his diet and taking bicarbonate of sodium after meals, he was enabled to follow his employment for the next six months, at the end of which time he was again seized with severe vomiting, apparently the result of catching cold. This attack lasted ten days and caused a loss of weight amounting to more than seven pounds. During the next eighteen months he suffered from six attacks of a similar nature, which produced such a degree of exhaustion that he was forced to give up his employment. Although he usually gained flesh between the attacks, he had nevertheless lost nearly two stones in weight since the ulcer healed.

On examination the patient was found to be very thin and somewhat anemic. The tongue was clean, the appetite fair, and thirst excessive. The thoracic organs were free from disease. The stomach was considerably dilated, the lower border of the viscus reaching nearly 3 inches below the level of the navel, but no peristaltic movements were visible. The right side of the epigastric region was tender on pressure, but no tumour could be detected. The superficial abdominal veins were dilated. The material vomited during the night was strongly acid, and contained an excess of free hydrochloric acid. In the early morning the stomach was found to contain about 12 ounces of a turbid fluid mixed with undigested food and mucus, the total acidity of which was 54. After filtration it digested egg albumen, and gave a positive reaction with phloroglucine-vanillin. When the vomiting was continuous the ejecta consisted entirely of alkaline and bile-stained mucus. The urine was much diminished in quantity, alkaline in reaction, and deposited phosphates on standing.

From the history and physical signs of this case we came to the conclusion that the ulcer had been situated in the pyloric region of the stomach, and during the process of cicatrisation had given rise to slight contraction of the orifice. As the result of the retention of food hypersecretion of the gastric juice had been excited, which in its turn had produced chronic gastric catarrh with intercurrent attacks of a more acute character. Daily lavage, combined with suitable diet and a course of carbolic acid and bismuth, soon relieved the pain and vomiting, and we subsequently learned that the patient gained considerably in weight, and, with the exception of an occasional attack of gastric catarrh, remained in fairly good health.

Examination of the stomach.—During the early stages of the complaint the abdomen is usually retracted and its walls rigid, but in long-standing cases the epigastric and umbilical regions are often distended from the presence of gas in the stomach and colon. Pressure over the site of the pylorus is always painful, but the localised tender spot in the epigastrium, which is encountered in cases of ulcer, is generally absent. If the patient is much emaciated, the peristaltic movements of the stomach are often visible through the thin parietes, and are seen to be especially energetic during an attack of pain. In every instance the stomach is dilated, and in advanced cases the lower border may reach several inches below the level of the navel. The position of the lesser curvature varies with the mobility of the pylorus. The superficial veins over the lower part of the abdomen are often dilated.

The existence of hypersecretion of the gastric juice is determined by investigating the contents of the stomach. When the organ is evacuated one hour after Ewald's test breakfast, the quantity of undigested food is considerably in excess of the normal. Bile is usually absent, and the material filters readily. The colour tests which indicate the presence of free hydrochloric acid are well marked, and on quantitative analysis the percentage of the mineral acid is found to be increased [·25—·4]. As the disease progresses, however, the secretion of acid usually diminishes, and in the final stages of the complaint it may be almost suppressed. A similar absence of acid is observed during recovery from acute gastric catarrh.

Exploration of the stomach in the early morning before breakfast shows that the organ contains a considerable quantity [5–20 ozs.] of an acid and often bile-stained fluid, which contains mucus and débris of food, and is capable of digesting albumen. Even when the stomach has been washed out on the previous night and no food has been taken in the interval, an active gastric juice can be evacuated on the following morning. In advanced cases, where chronic catarrh is the principal feature of the complaint, the contents of the viscus in the early morning consist of an alkaline bilious fluid mixed with mucus and saliva.

The physiology of digestion in hypersecretion.—By the employment of the tube the action of the abnormal gastric secretion upon the different food-stuffs can be accurately

determined. The presence of free hydrochloric acid in the stomach appears to prevent the conversion of starch into sugar by the saliva which has been swallowed with the food, and also the process of fermentation which favours the production of lactic acid. It is accordingly found that when the diet is composed entirely of carbohydrates very little digestion occurs in the stomach, and the filtered contents contain neither dextrin nor maltose. On the other hand, the proteid constituents of the food are more rapidly dissolved than under normal conditions, so that the period of gastric digestion is curtailed, and peptones are present in excess. In the later stages of the disease, when both the secretory and motorial powers of the stomach fail, the secretion of hydrochloric acid is so diminished as to render the digestion of the food a matter of considerable difficulty, while the enfeeblement of the muscular coat and consequent dilatation of the organ permit the stagnation and fermentation of the food, and give rise to the production of various gases and organic acids.

Diagnosis.—The principal point in the diagnosis of the disorder is the detection of an excess of gastric juice in the stomach in the early morning, even when the organ has been washed out on the previous night. This fact, combined with pain and vomiting a few hours after food, and the signs of dilatation of the stomach, renders the diagnosis of hypersecretion a matter

of certainty.

The only diseases with which it can be confounded are hyperacidity and chronic catarrh of the stomach. In the former complaint, however, the pain usually ensues within two hours of a meal, and subsides without either vomiting or diarrhea. Loss of flesh and anæmia are seldom observed, and the symptoms vary greatly in severity from time to time. The stomach is practically empty in the early morning. In chronic gastric catarrh pain is rarely a prominent feature of the case, but discomfort after meals, flatulence, and occasional acidity are frequent causes of complaint. The appetite is poor, and nausea and vomiting are present in the carly morning. The vomit consists principally of mucus and altered saliva, and if it occurs after meals the food shows little signs of digestion and free hydrochloric acid is usually absent.

Prognosis.—When the disease has become established the

chances of a permanent cure without recourse to an operation are somewhat remote. In addition to the dangers which arise from malnutrition the subjects of chronic hypersecretion are very prone to fall victims to pulmonary tuberculosis or to succumb to an intercurrent attack of acute gastric catarrh. Occasionally the scar of the former ulcer undergoes secondary ulceration, and fatal hæmatemesis or perforation ensues, or a perigastric abscess forms. In other instances peripheral neuritis or chronic disease of the kidneys develops, while in rare cases an attack of tetany, epilepsy, or acute paralysis of the stomach brings life to a sudden termination. These nervous complications are of considerable importance and will be con-

sidered separately in the next chapter.

Treatment.—In all cases the diet requires to be carefully regulated. The food should consist of nitrogenous materials and be taken in moderate quantities at rather more frequent intervals than in health. The fact that the digestion of starches is inhibited in the stomach indicates that such vegetables as potatoes, peas, beans, turnips, carrots, etc., should be employed sparingly, and farinaceous substances in general avoided as far Continental writers state that salt should be excluded from the food, as the ingestion of chloride of sodium helps to increase the production of hydrochloric acid, but we have never observed any bad effects from its use, nor any special benefit from its prohibition. Alcohol always disagrees and is apt to excite gastric catarrh; but when necessary a little hock or white wine diluted with some alkaline mineral water may be allowed with the meals. Cold water is usually the most efficient means of relieving the thirst, but when desired home-made lemonade, milk and soda water, or the 'Potus Imperialis,' may be used.

The chief indication for the employment of drugs is to relieve the pain and vomiting which ensue from the excessive acidity of the gastric contents. For this purpose full doses of bicarbonate of sodium, liquor potassæ, or calcined magnesia may be administered two hours after each meal, and repeated if necessary half an hour later. If the pain is exceptionally severe or obstinate in character, the alkali may be advantageously combined with tincture of opium, morphine, or codeine. In our own practice we are in the habit of using carbolic acid whenever the stomach is dilated, and have found that the

greatest benefit usually accrues from the action of that antiseptic. French physicians employ tincture of belladonna or the solution of atropine with the view of directly inhibiting the gastric secretion, but the use of these drugs has not been attended with success in our hands. The administration of large doses of the carbonate or subnitrate of bismuth (2 to 3 drachms) suspended in water, in the early morning, is often followed by considerable benefit. When the complaint is complicated by the coexistence of hysteria or neurasthenia a course of nervine sedatives and tonics, combined with massage of the trunk and extremities, may prove of use. In every case the bowels require to be regulated. The best aperient for this purpose consists of the artificial Carlsbad salts, of which two teaspoonfuls dissolved in half a pint of warm water may be given each morning before breakfast. Should these prove distasteful, the phosphate of sodium or the Rochelle salt may be employed. Aloes, colocynth, and other severe purgatives must be avoided on account of their tendency to excite catarrh of the colon

Whenever the stomach is dilated or medicinal treatment fails to relieve the symptoms, the organ should be thoroughly washed out each morning, the only contra-indication being the occurrence of hæmorrhage from secondary ulceration of the scar. As a rule warm water containing three grains of bicarbonate of sodium to the ounce is sufficient for the purpose, but some authorities prefer a weak solution of nitrate of silver (2 per cent.), sulphate of copper or sulphate of zinc (2 per cent.), or sulphate of alumina (5 per cent.), the patient being made to alter his position constantly during the operation, so as to bring the astringent fluid into contact with the whole of the inner surface of the stomach.

Should the patient continue to lose flesh and strength in spite of lavage and systematic treatment, the question of surgical interference must be considered. Carle has recently shown that even in cases of 'Primary Hypersecretion' the symptoms usually abate after the performance of gastroenterostomy; while in those in which the functional disorder ensues from cicatricial stenosis of the pylorus the results of pyloroplasty or of the aforementioned operation are extremely satisfactory.

During an attack of acute gastric catarrh the patient must

be confined to bed and hot poultices or turpentine stupes be applied to the epigastrium. No food should be permitted by the mouth, but if thirst is a troublesome symptom a small piece of ice may be sucked from time to time. The nutrition must be maintained by the use of nutrient enemata (p. 171). In cases of moderate severity a grain of calomel should be given every four hours until free purgation has been excited, after which the morphine and bismuth mixture may be administered three or four times a day, with a dose of Carlsbad salts or of phosphate of sodium each morning. As soon as the retching has ceased a cautious trial may be made with peptonised milk and soda water, bovril, or peptonised gruel, and if these can be borne the diet may be gradually increased. In the more severe cases rectal feeding is often necessary for a week or ten days, and the only means of checking the incessant vomiting is by the administration of hypodermic injections of morphine. Towards the termination of the attack retching may ensue from exhaustion, and under these conditions the injection of liquid nourishment into the stomach through a tube is sometimes of great service.

LITERATURE.

Reichmann, Berlin. klin. Wochensch., 1882, p. 606; 1884, p. 768; 1887, p. 199.
Bouveret and Devie, La Dyspepsie par Hypersécrétion Gastrique, Paris, 1891.
Bouveret, Maladies de l'Estomac, 1893, p. 161.
Jaworski and Gluzinski, Wiener med. Presse, 1886, p. 1681.
Honnigmann, Münch. med. Wochen., 1887, p. 951.
Riegel, Deut. med. Wochensch., May 25, 1892.
Van der Velden, Volkmann's Sammlung, 1886.
Schreiber, Deut. med. Wochensch., 1893, p. 692.
Ewald, Berl. klin. Wochensch., 1886, Nos. 48 and 49.
Korezinski and Jaworski, Deut. Arch. f. klin. Med., 1891, p. 578.
Hayem, Gaz. Hebdom. de Méd. et de Chirurgie, Aug. 1892.
Debove and Rémond, Les Maladies de l'Estomac, p. 152.
Martius, Deut. med. Wochensch., 1894, p. 638.
Voinoviteh, La Semaine Médicale, April 6, 1892.

CHAPTER IV

TETANY AND OTHER NERVOUS DISORDERS

[Tetany.—Tetanus.—Epilepsy.—Acute Gastric Paralysis]

In 1869 Kussmaul drew attention to the occasional occurrence of tetany in cases of chronic dilatation of the stomach, and since that time more than fifty instances of a similar nature have been recorded. 'Tetany of gastric origin' has conse-

quently become recognised as a clinical entity.

In the majority of the cases the gastrectasis has been found to be due to the presence of a chronic ulcer or its scar in the vicinity of the pylorus, but in a few instances the disease was either situated in the duodenum [Bamberger, Renvers, Dujardin-Beaumetz], or involved both the stomach and the first portion of the bowel [Loeb, Müller, Neumann, Thiroloix]. Cancerous infiltration of a simple ulcer has been noted three times [Bouveret and Devic, Riegel, Richartz]. Occasionally tetany is observed to ensue from dilatation of the stomach due to other causes than ulcer. Thus, in one of Müller's cases an hour-glass deformity of the stomach was present with twisting of the duodenum, while Blazicek has related one in which the pressure of an enlarged gall-bladder had given rise to obstruction of the first part of the duodenum. Among the rarer causes of gastrectasis atony of the stomach [de Baurmann], cancer of the duodenum [Trevelyan], and compression of the first part of the intestine by a cyst of the pancreas [Berlizheimer], have also been observed associated with tetany.

Symptomatology.—A careful consideration of the symptoms presented by the various cases appears to indicate that a tonic contraction of the muscles of the extremities is not the only feature of the nervous disorder, but that general convulsions of an epileptic or tetanic nature are also apt to supervene.

The nervous symptoms may therefore be divided into three classes:

- (1) A form of tonic contraction of the muscles of the extremities closely allied in its general features to true tetany.
- (2) An intermittent form of spasm attacking the muscles of the trunk, especially those of the jaw, neck, back, and respiratory system.
- (3) General convulsions of short duration attended sometimes by loss of consciousness, and resembling ordinary epileptic fits.

The two latter varieties never occur alone, but are always associated with the first-mentioned, which must therefore be considered as the fundamental type of the malady.

(1) Tonic spasm of the muscles of the extremities was the first symptom in every one of the recorded cases. It usually appears quite suddenly after a severe attack of vomiting or diarrhea, but it is sometimes preceded by a sense of numbness, tingling, or stiffness of the hands and feet. In typical cases the elbows and wrists are half flexed and the forearms strongly pronated; the fingers are drawn together and firmly bent over the thumbs, while the palms are hollowed by the approximation of the thenar and hypothenar eminences [The Accoucheur's Hand]. In the lower limbs the toes are bent downwards and adducted. The soles of the feet are hollowed, and the heels drawn up by the contraction of the muscles of the calves. Considerable pain is often experienced during continuance of the spasm, and in many instances the affected parts appear blue and are perceptibly cold to the The condition of the superficial reflexes is variable, but the deep reflexes are much exaggerated, and the muscles often react more readily than usual to the interrupted current. Sometimes an attack can be induced by percussing or stroking the skin of the epigastrium [Müller, Gerhardt], by the administration of an enema, by the passage of a stomach-tube [Collier, Fenwick], or by compressing the main artery of a limb.

The other phenomena associated with this condition are neither uniform nor of great importance. The pupils are often contracted during the attack, but they still react both to light and to accommodation. Severe headache is a frequent cause of complaint, and occasionally profuse perspira-

tions are observed. Retention of urine occurs in the majority of the cases, and when the fluid is drawn off by a catheter it is often found to contain traces of albumen. Sugar and acetone are occasionally detected in it [Biscaldi, Fenwick]. Cutaneous sensibility rarely undergoes any noticeable alteration, but in a few instances transient hyperæsthesia or anæsthesia has been observed. The pulse is full and regular, the breathing quick and shallow, and the face and extremities generally show signs of cyanosis. The temperature of the body is somewhat depressed at first, but in fatal cases it often rises, and may reach 109° F. before death [Collier]. The intellect often remains unaffected.

In almost every case the first attack is followed within a short time by several others, but occasionally the initial seizure is separated from the second by an interval of several days or even months. The actual duration of the spasm is also liable to considerable variation, lasting in some instances from five minutes to six hours, while in others it remains almost constant for three or four days. The general features of the disorder are well shown in the following case.

Case XLIX. A man, 34 years of age, was admitted into the London Hospital on February 26, 1893, suffering from severe vomiting, with eramps in the arms and legs. The patient was a carpenter by trade, and had enjoyed good health until six months previously, when he began to experience pain in the abdomen after meals, accompanied by nausea and vomiting. There was no family history of any importance. He had never been a heavy drinker, and had not suffered from syphilis. He stated that two days before admission [Feb. 24] he was suddenly attacked by violent vomiting, and a few hours later by eramps in the hands and legs which prevented him from walking.

On examination the patient was found to be a well-built and well-nourished man. The lips and checks were somewhat cyanosed, the pulse weak but regular, 80 per minute, the respirations 24 per minute, and the temperature 97° F. The arms were slightly abducted, the elbows and wrists flexed, and the fingers bent at the metacarpal and phalangeal joints. The palms were hollowed, and the thumbs adducted and covered by the fingers. The legs were rigidly extended, the toes flexed, and the feet inverted. Considerable pain was experienced when an attempt was made to alter the position of the limbs. The plantar reflexes and the knee-jerks were exaggerated. The bowels had not been opened for two days, nor had any urine been voided for the same period of time. On passing a catheter seven and a half

ounces of dark-coloured urine containing a trace of albumen were drawn off. No abnormal physical signs could be detected in the chest, and the heart-sounds were clear. The pupils were contracted, but reacted both to light and to accommodation. The fundi were healthy. The abdomen appeared to be somewhat distended, and was painful on pressure. The lower border of the stomach reached to the level of the navel, and a distinct splash was audible on palpation. Within the first few hours of his residence in the hospital, vomiting was frequent, the ejecta being bile-stained, sour-smelling, and considerable in quantity.

The following day the pulse was of fair volume and regular, 76 per minute. There were no signs of dyspnæa, but slight cyanosis was noticeable. The bowels had reacted to a dose of elaterium, but the patient still suffered from retention of urine. When the bladder had been emptied by a catheter, the fluid was found to contain a trace of albumen. The tonic spasm of the muscles still continued, and severe frontal headache was complained of. There was no alteration in the sensibility of the skin of the affected parts, and the intellect was quite clear. Vomiting was still troublesome. The next day (March 1) the cramps had disappeared, and the patient felt much better. In the evening, however, he was again attacked with severe spasm of the muscles of the extremities, preceded as on the former occasion by profuse emesis.

He also complained of twitching in the muscles of the face and mouth. The pupils were contracted, but reacted slightly both to light and accommodation. The knee-jerks and superficial reflexes were exaggerated. The skin over the outer side of the left thigh and leg was somewhat hyperæsthetic. The spasm lasted several hours and finally disappeared as suddenly as it had shown itself.

On March 4 the spasms recurred while the patient was under observation. The fingers of both hands were firmly flexed, and pressed over the thumbs. The legs became rigidly extended and adducted, while the feet assumed the position of equino-varus. The patient complained of great pain in the affected muscles, but there was no elevation of temperature nor any modification of cutaneous sensation. The attack subsided in about fourteen hours, and was followed by exhaustion and drowsiness.

The urine when voided was found to contain both albumen and sugar.

During the next ten days the patient enjoyed a respite from his disease. He could move his limbs easily and walk with comfort. The urine was passed in a voluntary manner and was quite normal.

On March 14 he was again seized with severe vomiting, and a few hours later the tonic spasms again made their appearance, and lasted for an hour and a half.

Three days later the tetany exhibited itself for the fifth time, preceded as usual by severe vomiting. This attack was accompanied by intense vertical headache, but only lasted for a short time.

From the 17th to the 31st of March the patient remained free from nervous symptoms. He complained, however, of pain in the epigastrium after meals, and on one or two occasions he vomited his food. The limbs were quite free from rigidity, and he could walk about the ward.

On April 4 vomiting recommenced, and several pints of bilious fluid were ejected during the course of a few hours. The tonic spasm of the extremities returned quite suddenly, and at the same time the patient complained of stiffness of the face and neck. The urine was found to contain a trace of albumen, when withdrawn by a catheter, and the pupils were contracted to the size of pin-holes. During the course of the next few hours the pain and stiffness in the muscles of the face increased, and the jaw became affected in a similar manner, so that great difficulty was experienced in swallowing. The respirations became jerky in type, the temperature rose to 102° F., and the patient succumbed to respiratory failure about twelve hours after the onset of his sixth attack of tetany.

At the autopsy the stomach was found to be considerably dilated, and in close proximity to the pylorus there was a chronic ulcer, which by its contraction had given rise to partial stenosis of the orifice. The brain and kidneys were apparently healthy, and no other visceral disease could be discovered.

Nearly 50 per cent. of the cases in which tonic spasm was the only symptom terminated fatally, death being often ushered in by delirium, a quick pulse, and a rapid rise of temperature, followed by coma with dilated pupils. In some instances the patient retained consciousness until the last, and succumbed to gradual failure of the heart.

In nearly one half of the cases the condition of simple tetany was complicated with the occurrence of convulsive seizures, which affected the muscles of the neck, jaw, back, and face. These attacks were intermittent and lasted from a few minutes to half an hour, disappearing as suddenly as they came, and leaving the muscles in a condition of semi-rigidity. During their continuance the patient was unable to open the mouth or to swallow, and in several cases opisthotonos was a marked symptom. This form of convulsions must therefore be regarded as a species of tetanus, and like the surgical variety of that disease its appearance was always

a sign of the deadliest import, for in every instance death ensued from failure of the respiration.

In about 12 per cent. of the entire number the initial tetany was said to have been followed by convulsions which were indistinguishable from those of ordinary epilepsy. The fits were repeated in rapid succession, and a fatal termination was recorded in two-thirds of the eases.

Etiology.—The eause of these nervous phenomena has been the subject of much discussion. Kussmaul originally suggested that they were due to an excessive loss of fluid from the eireulation produced by the vomiting, and although this theory was subsequently diselaimed by its author it has received the support of Fleiner and Jürgensen. Blazicek, however, has shown by investigation that there was no actual loss of water from the blood in his ease, nor is tetany a common symptom of eholera and other diseases in which large quantities of fluid are drained away from the body. The fact that the convulsions may often be induced by the passage of a stomach-tube or by irritation of the skin of the epigastrium appears to suggest that they might possibly be due to reflex eauses. The presence of entozoa in the intestine in the ease described by Riegel and the disappearance of the complaint after the evacuation of the parasite also seem to support this view. It must be remembered, however, that in ordinary eases of tetanus peripheral stimulation will usually provoke a spasm, while in tetany the tonic contraction of the extremities may often be induced by exposure to cold or by compressing the main artery of a limb.

The theory which enjoys the widest acceptance attributes the nervous symptoms to the absorption into the general circulation of some organic poison produced in the dilated stomach by bacterial action (auto-intoxication). According to Bouveret and Devie the connecting link between the two affections is to be found in the excessive secretion of hydrochloric acid which usually accompanies chronic ulcer of the stomach. These observers were able to separate from the gastric contents of one of their cases a substance that was soluble in alcohol, and which gave rise to convulsions when injected into animals. Fleiner is also stated to have obtained somewhat similar results. On the other hand, Jakseh and Berlizheimer, Müller, and Blazicek all failed to obtain a specific poison from the cases under their care, while Gumprocht's claborate investiga-

TETANY AND OTHER NERVOOS PASSENDORS SPELL!

tions were also negative in their results. This latter writer points ont that whenever an organic poison is absorbed from the gastro-intestinal tract a certain proportion must be eliminated by the kidney, and he was able to demonstrate in one case that the renal secretion possessed an abnormally high protoxic coefficient, which, however, continued both during the attacks and in the intervals. Ewald and Jacobson extracted a body allied to ptomaine from the urine in one instance.

With regard to the possible influence of hyperchlorhydria it may be noticed that an excess of acid was absent in the case recorded by Blazicek, while tetany has been observed in pyloric obstruction due to cancer and external pressure, conditions which are not usually accompanied by excessive acidity of the gastric juice. While, therefore, it is highly probable that the nervous symptoms are due to auto-intoxication, there is at present no proof that hydrochloric acid is an indispensable factor in their production.

Another point of interest in connection with the etiology of the complaint is the occurrence of albuminuria. Both Blazicek and Hoffmann have recorded cases in which the kidneys presented signs of Bright's disease after death, but in others the organs appeared normal to the naked eye. In the case which has been cited at length, the albuminuria appeared only during the attacks, and was probably the result of congestion of the kidneys consequent upon the failure of the respiration. It is possible, however, as Trevelyan has suggested, that it may sometimes be due to the excretion by the kidneys of the poisons absorbed from the stomach. In any case, a disordered state of the renal functions, whether it has been brought about by previous disease or by the irritation of poisonous products, would necessarily retard the elimination of the toxins, and thus increase their effect upon the nervous centres. The occasional appearance of sugar in the urine is probably devoid of any clinical significance, notwithstanding the fact that Biscaldi attributed the tetany in his case to the presence of acetone in the blood.

According to Gumprecht, nearly three-quarters of the cases of gastric tetany occur between the months of January and March, and there can be no doubt that the disorder is exceptionally rife during the cold months of the year. The greater liability of men to the disorder obviously depends upon the

greater frequency of chronic ulcer of the pylorus and duodenum in the male sex.

Prognosis.—Tetany is always a very serious complication of gastric ulcer, and about 75 per cent. of the cases terminate fatally. It is rare for the first attack to prove fatal, but in Marten's case death occurred in four hours, and in that reported by Trevelyan in about six. More often the seizures succeed each other at short intervals, and finally give rise to paralysis of the respiratory muscles. The special signs which betoken the approach of death are delirium, a rapid rise of temperature, and deepening coma with dilatation of the pupils. Although the mortality is apparently very high, we believe that slight attacks of tetany are far more common than is generally believed, and that under proper treatment the mild form of the complaint is capable of cure. In this connection the following case presents some features of interest.

Case L. A gardener, 46 years of age, was admitted into hospital in 1888, suffering from chronic dilatation of the stomach. It appeared from his history that he had always enjoyed excellent health until the age of 42, when he began to suffer from pain in the epigastrium after food, which was sometimes followed by vomiting. For three years these symptoms became gradually aggravated until he was unable to swallow any form of solid food without severe pain, and was accustomed to vomit several pints of sour fluid once or twice a week. The immediate cause of his seeking medical advice was his intense dread of experiencing a return of what he termed 'the convulsion spasms.' It seemed that during the recent cold weather he had been attacked two or three times by sudden and severe vomiting, and almost immediately afterwards the hands and feet had become stiff and painful, and he had suffered from cramps in the calves of the legs. He stated that the hands were clenched with the thumb inside, the feet drawn down, and the toes bent towards the sole. The spasms lasted several hours on each occasion, and left the limbs sore. During the attacks he could not walk.

On examination the patient was found to be extremely thin and anæmic. The heart-sounds were weak but clear, and the lungs presented no signs of disease. The stomach was greatly dilated, and its lower border extended nearly three inches below the umbilicus; a loud splash was audible on manipulation, while pressure over the pyloric region caused pain. There was no tumour to be felt. The bowels were obstinately confined, and the temperature of the body was slightly below the normal. The urine contained neither

albumen nor sugar. Partial stenosis of the pylorus, due to the contraction of a chronic ulcer in its vicinity, was diagnosed, and the stomach was accordingly emptied by means of a soft tube, and the organ thoroughly washed out. Under this method of treatment, combined with careful dieting, the patient rapidly improved, and finally left the hospital after having learnt to perform lavage for himself. In May, 1892, he again made his appearance. He stated that since his discharge from the hospital he had continued to use the stomach-tube several times a week, with the result that the pain and vomiting had been less severe than formerly, and the spasms had never troubled him again. The stomach was still dilated, but the patient had gained several pounds in weight. For the last two years there has been no news of him.

Although the notes of this case are very incomplete, inasmuch as the nervous symptoms were not observed by a medical man, there can be little doubt that the so-called spasms were in reality closely akin to those previously described, a recurrence of which it is probable the treatment by lavage had tended to prevent.

In cases where the tonic spasm is complicated by seizures of a tetanic or epileptiform character, the prognosis is hopeless. An excess of albumen in the urine, indicating the previous existence of renal disease, is a sign of evil omen.

Treatment.—The first indication is to wash out the stomach and to procure a thorough evacuation of the bowels. Warm water is quite sufficient for the purpose, and antiseptic solutions should be avoided. If vomiting has been severe, a pint of sterilised salt solution may be left in the stomach at the conclusion of the operation, or a hot saline enema may be administered. A full dose of calomel is usually the best purgative. Boas recommends the use of diuretics, and we believe that the employment of a hot air bath as in cases of uramia may sometimes prove of value. Many writers urge the prohibition of alcohol, but if signs of failure of the heart are present, ether, ammonia, strychnine, or some other cardiac stimulant, may be given, either by subcutaneous injection or in the form of an enema. There can be no doubt that the only way to prevent a recurrence of the complaint is to remove the pyloric obstruction, either by the performance of pyloroplasty or gastroenterostomy.

Acute Paralysis of the Stomach has not hitherto been recognised as a result of chronic ulcer. It is true that in one of Fagge's original cases a sloughing abscess was present behind

the duodenum and was found to communicate with the bowel, but its mode of origin was not explained. We have seen two cases of gastric ulcer in which the sudden development of acute dilatation of the stomach appeared to be the immediate cause of death. The first instance resembled in its general features the ordinary type of the disease, sudden and profuse emesis, with anuria, exhaustion, and enormous dilatation of the stomach, bringing life to an end in about forty-eight hours. In our second case the disease was preceded by tetany, and is therefore worthy of a more detailed notice.

Case LI. A lady began to suffer at the age of 29 from pain after meals, which gradually increased in severity until at the end of about twelve months epigastric pain and vomiting ensued after every attempt to partake of solid food. She had lost a great deal of flesh, and was much exhausted. These symptoms subsided after rest in bed and liquid nourishment for three weeks, and she appeared to be gaining strength, when she was suddenly attacked by a tonic spasm of the hands and arms, accompanied by a low form of delirium. The attack was preceded by vomiting and accompanied by retention of urine. When we first saw her the spasm had disappeared, but she was unable to concentrate her thoughts and answered the questions put to her somewhat at random. There was no pain or vomiting, and the urine was passed naturally and contained neither sugar nor albumen. The skin was dry, the pulse slow, and the temperature slightly raised above the normal. The thoracic viscera appeared to be healthy, and there was no pigmentation of the skin or mucous membranes. The lower border of the stomach reached within half an inch of the level of the navel, and there was a localised tender spot in the centre of the epigastrium. There had never been any hæmatemesis. Under careful dieting these symptoms gradually subsided, and the patient began to gain strength.

About six weeks later, however, she was suddenly seized with violent vomiting, accompanied by cramps of the limbs. When we saw her on the following day there was slight stiffness of the hands and arms, the fingers being somewhat adducted and bent over the thumbs. There was complete retention of urine, and the patient was so exhausted that she could hardly turn over in bed. The pulse was quick, the tongue foul, and the temperature subnormal. The vomiting was incessant, and despite the fact that she had taken no food for more than twenty-four hours, large quantities of fluid having a slightly bilious tinge were vomited every hour or two without apparent effort. On examination the stomach was found to be enormously dilated, and practically to occupy the whole of the anterior aspect of

the abdominal cavity; but no peristaltic movements could be detected. Despite the excessive vomiting the organ still appeared full of fluid. An attempt was made to pass a soft tube, but it gave rise to severe retching, and fatal syncope occurred about an hour afterwards.

Although it was impossible to verify the diagnosis by an autopsy, the clinical features of the case strongly indicated the existence of chronic gastric ulcer with acute paralytic distension of the stomach. The pain and vomiting after solid food, which had existed for more than a year, combined with the tender spot in the epigastrium and the rapid improvement which followed confinement to bed and restriction to a liquid diet, were highly suggestive of ulcer, while the fact that the stomach was normal in size six weeks before death, along with the sudden onset of the symptoms and paralytic condition of the organ, seemed to indicate that the dilatation had taken place very rapidly.

The chief point of interest in the case is the association of tetany with a paralytic condition of the stomach. It is well known that acute gastric dilatation occasionally occurs in diphtheria, influenza, enteric fever, and other diseases in which toxins accumulate in the blood, so it is reasonable to suppose that the paralysis of the stomach owes its origin to the action of these poisons upon the nervo-muscular apparatus of the organ. In cases of tetany the spasms are almost invariably preceded by profuse vomiting, the quantity of fluid which is rejected being out of all proportion to the amount that has been swallowed. It is also worthy of notice that during the attack peristaltic movements of the stomach are seldom observed, even if they were previously present, while in most instances paralysis of the bladder, with retention of the urine. is a concomitant symptom. These facts suggest that in many cases of gastric tetany acute paralysis of the stomach, and perhaps of other hollow viscera, may precede the spasmodic seizures, and constitute an independent symptom of the toxic poisoning.

LITERATURE OF GASTRIC TETANY

Bamberger, Virchow-Hirsch Jahresbericht, 1892, p. 174. Berlizheimer, Berl. klin. Wochen., 1897, p. 773. Blazicek, Wien. klin. Wochen., 1894, p. 326.

Bouveret & Devic, Revue de Médecine, Feb. 1892. Collier, Lancet, 1891, i. p. 1251. De Baurmann, Bull. Soc. Méd. des Hôpitaux, 1889, p. 166. Dreyfus-Brissac, Gaz. Hebdom., 1885, No. 27, p. 439. Dujardin-Beaumetz, L'Union Médicale, vol. xxxvii. p. 169. Fenwick, Clin. Soc. Trans., vol. xxviii. p. 13. Gassner, Thèse de Strassbourg, 1878. Gerhardt, Berl. klin. Wochen., 1888, p. 74. Gumprecht, Centralbl. f. inn. Med., 1897, p. 569. Hanot, Lèprevotte. Thèse de Paris, 1880. Hayem & Gaillard, Gaz. des Hôpitaux, 1883, p. 900. Hoffmann, Deut. Archiv f. klin. Med., 1888, p. 603. Jürgensen, Deut. Archiv f. klin. Med., 1898, p. 327. Kussmaul, Deut. Archiv f. klin. Med., 1869, p. 455. Leven, Maladies de l'Estomac, Paris 1879. Loeb, Deut. Archiv f. klin. Med., 1889, p. 95. McKendrick, Lancet, 1898, ii. p. 796. Marten, Laneet, 1887, i. p. 74. Merlin, La Loire Médicale, Nov. 1890. Müller, Charité-Annalen, 1889, xiii. p. 309. Nason, Lancet, 1891, ii. p. 44. Neumann, Deutsche Klinik, 1857. Paliard, Revue de Médecine, 1888, p. 406. Renvers, Berl. klin. Wochen., 1884, p. 74. Sievers, Berl. klin. Wochen., 1898, p. 680. Thiroloix & Du Pasquier, Bull. Soc. Anatom., series v. vol. 7. Thomson, Ann. Univ. Med. Sciences, 1888, p. 381

Trevelyan, Lancet, 1898, ii. p. 791.

CHAPTER V

PERIGASTRIC AND PERIDUODENAL ABSCESS

[To be read with pp. 58-66]

Although a localised abscess in the abdomen had long been recognised as an oceasional result of perforation of the stomach, the clinical features of the complaint do not seem to have attracted much notice until the year 1845, when a case was successfully diagnosed by Drs. Barlow and Wilks. In 1879 Leyden published his celebrated paper upon 'Pyo-pneumothorax subphrenicus,' and since that time the symptoms and signs of a perigastrie or, as it is sometimes called, subdiaphragmatic abscess have received a considerable amount of attention. The following remarks concerning the clinical aspect of the disease are based upon an analysis of fifty-six cases, which include twelve of our own.

Previous history.—In almost every instance the patient has suffered from the symptoms of gastric ulcer for a considerable time before the occurrence of perforation. In only 8 per cent. of our cases had pain after food existed for less than a year, while in the remainder it had lasted from one to five years, or even longer. As a rule there have also been acidity, vomiting, constipation, flatulence, and localised tenderness in the upper part of the abdomen. On the other hand, it is important to observe that the victims of perigastric absects have seldom suffered from hamatemesis, only about 6 per cent. of our cases having vomited blood before the perforation took place.

It sometimes happens that the previous symptoms of ulceration have been so slight as to escape the patient's recollection (case 52), and it is only after persistent questioning that the former existence of some painful disorder of the stomach can be determined. As this fact is always of the

greatest importance in the diagnosis, no pains should be spared to obtain a correct history.

Initial symptoms.—In the great majority the occurrence of perforation is accompanied by characteristic symptoms. In almost every instance the patient is seized with sudden and severe pain in the abdomen, which in about 28 per cent. is followed by collapse. Occasionally retching and vomiting or an attack of diarrhœa are prominent symptoms, or a succession of rigors may mark the commencement of the peritoneal inflammation. For the purposes of description it is convenient to recognise two varieties of perigastric abscess, the acute and the chronic. The former is most often encountered in young women suffering from a chronic ulcer near the cardiac orifice, in whom the perforation is apt to excite suppuration beneath the left wing of the diaphragm. In such an abdominal tumour is usually absent, and the principal symptoms depend upon secondary inflammation of the thoracic viscera. In the chronic variety, which occurs chiefly in men of middle age, the symptoms gradually subside as the abscess becomes encysted by fibrous adhesions, and many months or even years may elapse before the appearance of a tumour or the supervention of pleurisy or pneumonia demonstrates the real nature of the malady.

(1) The Acute Form.—As a rule the symptoms of gastric perforation are very conspicuous, but if the patient is the subject of tuberculosis, cancer, or of some other debilitating malady, the accident may only be portrayed by an increase of the abdominal pain or by sudden collapse. Several interesting cases of this description have come under our observation.

Pain is invariably complained of as soon as the initial shock has passed off, and is chiefly referred to the epigastrium and left hypochondrium. It is usually increased by deep inspiration, coughing, and by movements of the body, and is sometimes so severe as to necessitate the administration of morphine. Dyspnæa is another frequent symptom, the breathing being quick, shallow, and costal in type. In every instance the patient exhibits the appearance of extreme illness, and usually lies upon her back with the head low and the knees bent; but when pulmonary or cardiac complications ensue she often prefers to be propped up in bed. Cough is almost always present after the first few days, and is occasionally accompanied by rusty expectoration. Retching and vomit-

ing are troublesome symptoms in many cases. The bowels are usually confined. The pulse is quick, small, and feeble; the urine is retained or micturition is difficult, and incessant hiccough is often present. In the majority of cases the formation of the abscess is accompanied by a remittent form of fever, the temperature rising to $102^{\circ}-103^{\circ}$ F. at night, and falling to $99^{\circ}-100^{\circ}$ F. in the morning. It is important to notice, however, that in debilitated persons, and also in those who have been subjected to laparotomy for gastric perforation, a large abscess may form beneath the diaphragm without any rise of temperature. In the following case continued fever without obvious cause was the only symptom which led to the suspicion of a perigastric abscess.

Case LII. A young woman was admitted into the London Hospital under the care of the obstetric physician. She had been apparently in good health until a few weeks previously, when she was supposed to have had a miscarriage. She made no complaint of pain, but her temperature was elevated, the pulse quick, and she felt weak and ill. The uterus and the pelvis were examined, but no disease could be discovered, and as careful inquiry seemed to negative the supposition of abortion she was transferred to the care of the physician. There was no cough, expectoration, or other pulmonary symptom, and the thoracic organs were normal. The back of the right lung was carefully examined, lest there should be a localised empyema, but no physical signs of that disease could be detected. The abdomen was apparently normal, and there was neither albumen nor sugar in the urine. At first she denied that she had ever suffered from pain after food, but she afterwards admitted that for some months before her illness she had been subject to 'indigestion.' She never had During her residence in the hospital she never hæmatemesis. complained of pain after food, and there was no vomiting or flatulence. Nevertheless the temperature continued markedly remittent in character, rising to 103° and 104° F. in the evening, and falling to normal in the morning. As the pyrexia appeared to indicate the presence of an abscess which could not be discovered, it was surmised that there might be suppuration behind the stomach in connection with an old ulcer. No operation was considered advisable, and the patient gradually sank from exhaustion.

At the autopsy the organs of the body were found to be in a healthy condition except the stomach, on the posterior wall of which was a chronic ulcer that had perforated the viscus and communicated with a large abscess situated in the cavity of the lesser omentum.

The above case shows how difficult it may be to diagnose a perigastric abscess, even when the collection of pus is a large one. The patient's own impression was that her illness had resulted from a miscarriage, and it was only after careful examination that the obstetric physician came to the conclusion that the pelvic organs were not responsible for the symptoms. There was no abdominal pain, vomiting, or tenderness, nor was there any history of hæmatemesis, and it was only after persistent questioning that the patient confessed to having previously suffered from 'indigestion.' No distension of the abdomen or tumour was observed, and if it had not been for the persistent pyrexia and weakness no idea of suppuration behind the stomach would ever have been entertained.

Rigors occur in about 10 per cent. of the cases, but they are seldom frequent unless pylephlebitis or general pyæmia has resulted from the abscess.

In the great majority of the cases secondary inflammation of the pleura or lung supervenes within a few days after the perforation, and often masks to a great extent the abdominal symptoms. The average duration of the disease is about a fortnight, but if the pus perforates the diaphragm or bursts into the peritoneal cavity death may occur at a much earlier period. In the following case the complaint ran a very acute course, and terminated by general peritonitis on the sixth day.

Case LIII. A nursemaid, aged 23, was admitted into the London Hospital for pain in the abdomen and vomiting. She stated that for the last eighteen months she had suffered from pain and sickness after meals, but had never vomited any blood. Her present illness began two days before, when about one hour after dinner she felt as if something had given way in the abdomen; she was immediately seized with an agonising pain and became partially unconscious. When she revived she complained of intense pain all over the upper part of the abdomen, and had several attacks of shivering followed by sweating. There was also much sickness.

On admission the patient was found to be very anæmic, and looked extremely ill. The respirations were quick and shallow, the pulse 112 per minute, and the temperature 101.5° F. Great pain was complained of over the left side of the chest and in the hypochondrium, which was increased by inspiratory efforts and movement of the body. Examination showed the abdomen to be somewhat distended and resonant on percussion, with tenderness on palpation

over its upper part, but no tumour could be detected. Signs of acute

pneumonia were present in the lower lobe of the left lung.

Two days later a loud friction sound could be heard over the lower half of the left chest. The temperature rose to 103° F. at night and fell to 100·5° F. in the morning, and retching ensued after every attempt to administer food. On the sixth day of the disease the whole of the abdomen was distended, and tympanitic on percussion. The apex of the heart was in the fourth intercostal space in the nipple line, and a pericardial friction sound was heard over the lower part of the organ. At the base of the left chest there was dulness on percussion with diminished respiration, and about six ounces of turbid serum were evacuated on aspiration. A few hours later the patient suddenly became collapsed and died.

Autopsy.—The abdomen contained several ounces of pus, and the coils of intestine showed evidence of recent peritonitis. There was a large abscess cavity between the left lobe of the liver, the diaphragm, and spleen, with recent adhesions between the stomach and the abdominal wall. These latter had apparently ruptured and allowed the pus to find its way into the general cavity of the peritoneum. On the anterior wall of the stomach, about one inch from the lesser curvature and close to the esophageal opening, was a perforated chronic ulcer almost the size of a shilling. The base of the left lung was in a condition of grey hepatisation, and its pleural surface

covered with lymph. There was also recent pericarditis.

(2) The Chronic Form.—In this variety the leakage is probably very slight and confined to the immediate neighbourhood of the perforation, while the pus becomes gradually encapsuled by thick fibrous tissue, which prevents its further diffusion. In all cases the initial symptoms are identical with those of the acute disorder—sudden and severe pain in the abdomen, with dyspnæa, continued fever, and occasional rigors or attacks of vomiting. Instead, however, of running a rapid course and exciting secondary inflammation of the thoracic organs, the symptoms gradually subside and the temperature falls to the normal, and it may not be for many months or even years that the supervention of pleurisy, perforation of the diaphragm, or the appearance of a tumour upon the side of the chest demonstrates the presence of the abscess.

As a rule, however, the patient continues to experience pain in the abdomen during the whole course of the complaint, and when this is constant and accompanied by loss of flesh and vomiting, it is apt to excite the suspicion of malignant disease of the stomach. Rigors are occasionally observed, and profuse sweating at night is a frequent symptom. The appetite is lost, the bowels are confined, and great weakness is usually present. The following case is a good illustration of the general features of the complaint.

Case LIV. A man, aged 38, was admitted into the London Hospital on January 31, 1881. He was a weaver, and had enjoyed good health until 'some time ago,' when he began to suffer from indigestion, the food appearing to lie heavy on his stomach and producing great pain. He stated that he had suffered from an injury to the back, but its nature and the date are not mentioned in the notes, and he evidently did not attach any importance to it. He had never had syphilis.

On admission he complained of very severe pain in the left hypochondrium, which extended backwards round the left side as far as the spine. All this region was very tender on percussion, the most tender spot being opposite the tenth and eleventh ribs. He was unable to lie on his right side, so that he constantly reclined on the left, with the neck bent forwards and the knees drawn upwards. Pulse 90; temperature 99° F. He had no appetite; the tongue was thickly furred, and the bowels obstinately confined. The liver dulness extended from the fifth costal interspace to one inch below the ribs, and the edge was slightly tender on pressure. In the left hypochondrium there was dulness on percussion, which extended upwards to the heart's apex and downwards to the level of the umbilieus. The heart's apex was not raised, and there were no murmurs. The lungs were healthy.

On February 10 he had constant vomiting, and complained of the severity of the pain, which extended from the left hypochondrium to the spine, and was increased directly he attempted to take any food. The recti museles were hard and rigid. On the left side near the spine there was dulness on percussion, but no increased pulsation, and no murmur. He had constant nausea.

Feb. 15.—A hardness could be felt below the left hypochondrium, and there was a dull space on percussion in the left hypochondrium, measuring three inches longitudinally by six laterally (see fig. 52). The lower border of the liver was also remarked searcely to descend on full inspiration, and there was some dulness on percussion, and increased resistance to pressure immediately above the umbilieus. The man continued in much the same state until April 22. The pain was excessive and constant, and required the frequent use of subcutaneous injections of morphine. He steadily lost flesh, and was then only 6 st. 12 lb., having weighed 8 st. on admission. The temperature varied from 98° to

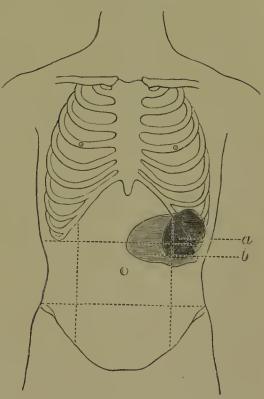
100° during February, but rose to 102° in March, and afterwards oscillated until April 22, at which date it rose to 103°, and the patient complained of great pain at the base of the lungs behind, although neither dulness nor friction-sounds could be discovered.

April 27.—He had a severe rigor, the temperature rising to 106° F.; but the pain in the left side was not so severe as usual. The left chest was now found to be dull on percussion, with absence of breath-sounds, and diminished vocal and tactile fremitus, but no friction-sounds could be heard.

April 29. — He had severe sweatings; the area of dulness was considerable in the left hypochondrium, but the tenderness was not increased.

Friction-sounds were first discovered on May 2 near the apex of the heart; rigors recurred from time to time, and an herpetic eruption showed itself on the lips, palate, and roof of the mouth. After this period the appetite began to improve, the pain decreased, and the fever disappeared.

May 12. — Frietion-sounds could be heard over the whole of the left hypo-ehondrium, where there was formerly such marked dulness on percussion. The dulness at the back of the lung had much diminished, and the pain only came on after food.



on percussion. The dulness Fig. 52.—Diagram of a case of perigastric at the back of the lung had abscess. a, part dull on percussion; b, part comparatively dull on percussion.

From this time he rapidly improved; the pain entirely left him, the appetite returned, and he gained (so the notes state) as much as six pounds in weight in a single week.

June 22.—When he was discharged at the end of five months, the percussion note over the left base was normal, and no friction could be detected. There was some fulness under the left hypochondrium, but the part was resonant on percussion.

There is, of course, always a certain amount of doubt about a case in which it is impossible to verify the diagnosis by a

post-mortem examination, but when the course and termination of the preceding case are taken into consideration the existence of a perigastric abscess must be considered almost certain. The patient had suffered from the symptoms of gastric ulcer for a long time before he entered the hospital. On his admission, the pain, although constant, was still increased by food, and beneath the left hypochondrium there was an illdefined tumour, which was very tender on pressure and dull on percussion. After suffering great pain for three months, severe rigors ensued, followed by the signs of acute pleurisy with effusion in the left side of the chest, after which the tumour disappeared and the case terminated in recovery. At first it was considered possible that the disease might be cancer of the stomach, but the continued fever and the subsequent progress of the case soon negatived that supposition. That the tumour was not due to abdominal aneurism was shown by the absence of pulsation and murmur, and by its subsequent disappearance; while its rapid development, its peculiar position, and its association with severe gastric symptoms eliminated the possibility of an hydatid cyst. It is most probable, therefore, that a small abscess had originally formed near the upper curvature of the stomach in connection with a chronic ulcer, and had subsequently burst into the left pleural cavity.

In some instances the pain continues for a long time after perforation has taken place, and is accompanied by inflammation of the thoracic viscera, and finally by the evacuation of the abscess through one of the lower intercostal spaces. The following was probably a case of this kind.

Case LV.—A gentleman, 45 years of age, who consulted us in 1898, gave the following history. In the year 1888 he began to experience severe pain in the epigastrium after meals, with vomiting and constipation. About two years later he suddenly felt something give way in the stomach while engaged in athletic exercise, and was immediately attacked with violent pain and collapse. After many months' illness he gradually recovered, but continued to experience pain after food. In 1893 he suffered from acute pleurisy on the right side which laid him up for several weeks, and subsequently he suffered from occasional attacks of severe pain in the right side of the chest which were supposed to be due to gall-stones. In 1894 a tumour appeared at the posterior and lower part of the right chest. When it was opened a quantity of curdy pus was evacuated, and the opening continued to discharge for more than two months. Towards the end

of the year he vomited a large quantity of blood, and almost died from a recurrence of the hæmorrhage. Since his recovery he had remained very weak, and had suffered from periodic vomiting and loss of flesh.

On examination the patient was found to be extremely emaciated. When he lay upon his back a curious deformity of the abdomen was observed. The left hypochondriae, umbilical, and lumbar regions were occupied by a large swelling, over the surface of which peristaltic movements were distinctly visible. The tumour was tympanitic on percussion, and a loud splashing sound was elicited on manipulation. The other side of the abdomen appeared quite collapsed and empty, and the bony structures at the back of the cavity could easily be felt. There were thickening and adhesion of the pleura on the right side, and an old sear between the tenth and eleventh ribs posteriorly. The shape of the abdomen was not altered by position, and was very evident in the erect posture. The other organs of the body were normal. The contents of the stomach contained an excess of hydrochloric acid.

The diagnosis in this case may be regarded as doubtful, but a little consideration will show how strong are the grounds for regarding it as one of perigastric abscess. After suffering for a long time from the symptoms of gastric ulcer, the patient was suddenly attacked with severe abdominal pain and collapse, and afterwards had persistent pain in the right hypochondrium. Subsequently an abscess pointed at the back of the chest, and continued to discharge for some months. The only rational explanation of such a succession of phenomena seems to be that perforation of the stomach or first part of the duodenum had occurred, and set up a small intraperitoneal abscess, which gradually increased in size and made its way between the liver and the right wing of the diaphragm.

The chief difficulty is to understand how, if the perforation took place in 1890, the abscess did not make its appearance until 1894. It will be observed, however, that during the whole of this period the patient was never free from pain, while the violent attacks from which he occasionally suffered, and the acute pleurisy on the right side, showed that some kind of inflammatory mischief must have existed beneath the diaphragm. We can only suggest that the abscess soon became encysted, and remained latent during the greater part of the time. That this may occur is proved by the following

case, which in most of its features was very similar to that just related.

Case LVI. A woman, aged 39, was admitted into the Worcester Dispensary on March 6, 1828, under the care of Dr. Streeton. She was suffering from great pain and tenderness in the epigastrium and right hypochondrium. She had also a cough with muco-purulent expectoration, and there was a hard painful tumour on the back, just below the angle of the right scapula.

She was said to have enjoyed fairly good health till about four years previously, when she had severe pain in the right side, for which she was under treatment five months, being relieved, but never

entirely cured.

Shortly after her admission the above-mentioned tumour broke and discharged purulent matter, and for some time afterwards she appeared to improve, though a sinus remained. At the end of June a second swelling, situated in the seventh intercostal space, about an inch and a half anterior to the angle of the ribs, opened and discharged pus and blood. In the first week of July the gastric contents began to pass through this fistula almost immediately after fluid had been swallowed. Milk escaped in a curdled condition five minutes after it had been injected. The patient died at the end of the month.

Autopsy.—Strong adhesions were found between the right lung and the thorax, and between the lung and the upper surface of the liver, the diaphragm being lost in a mass of 'fibro-cartilaginous' tissue. There was extensive phthisis. In consequence of the abovementioned adhesions the stomach and duodenum were so much displaced that the pyloric end of the stomach passed upwards between the right and left lobes of the liver, the pylorus being attached to the upper surface of the organ and the cartilages of the eighth and ninth ribs on the right side.

The duodenum passed with a sharp bend downwards, also between the two lobes of the liver, being much constricted thereby, and presented just at the bend a perforation which communicated with the second of the sinuses mentioned above, by a track behind the ribs two and a half inches in length. The sinus lay in the tough fibrous tissue which connected the liver with the chest wall. The first sinus appears to have communicated with the cavity of the thorax, though it is said that 'there was no appearance of disease on the pleura pulmonalis corresponding with the opening.'

In the next case the appearances of the abscess after death showed that it must have existed for a considerable time, and might have continued much longer had it not excited fatal inflammation of the pleura.

Case LVII. A man, aged 51, was admitted into the London Hospital for pain in the chest and debility. He stated that, two years before, he had vomited blood and had since suffered from pain in the abdomen. For the last week the pain had been constant and increased by inspiration. At the commencement of his illness some accident had happened, but he could give no details as to its nature. The patient was obviously very ill. The breathing was quick and shallow, and great pain was complained of in the epigastrium and left side of the chest. The upper part of the abdomen was rigidly contracted, and very tender upon pressure. A friction sound was heard over the back of the left lung. Death occurred in a few hours.

Autopsy.—There was a large chronic ulcer on the lesser curvature of the stomach near the esophageal opening, which had perforated all the coats of the organ. The upper border of the stomach was firmly adherent to the left lobe of the liver, and when the organ was removed an abscess cavity was found which was bounded above by the diaphragm, on the left by the diaphragm and spleen, below by the stomach, and on the right by the liver. The walls of the sac were composed of dense fibrous tissue, and were separated from the ulcer by fibroid material nearly an inch in thickness.

In the next case pleurisy with effusion ensued a few weeks after perforation, and it was not until about five months later that the abscess pointed in the left hypochondrium.

Case LVIII. A lady, 25 years of age, who had suffered from pain after food and vomiting for nearly two years, was attacked, when running downstairs, with violent pain in the abdomen, followed by vomiting and collapse. There was great tenderness over the epigastrium, and the temperature rose to 102° F.; but during the course of the next week the pyrexia disappeared. At the end of three weeks the pain became greatly increased, and when we saw her at the end of the month there was considerable effusion of fluid into the left chest, and the epigastrium was somewhat distended and tender upon pressure. No tumour could be detected. The pleurisy gradually subsided, but she still complained of much pain in the left side of the chest, more especially at night, and had occasional attacks of fever. About five months after the onset of her illness a hard swelling made its appearance in the left hypochondrium, which increased in size and gradually approached the surface. When it was opened a large quantity of pus was evacuated, and the cavity eventually closed.

These facts appear to indicate that under certain conditions perforation of the stomach may give rise to a small abscess which remains strictly circumscribed for a considerable length of time.

Abdominal Abscess is a rare result of the perforation of a duodenal ulcer, but we have been able to collect the details of twenty-two cases of this description, including three of our own. In every instance the ulcer was of long standing, and the proportion of males to females was 10 to 1. In the great majority the formation of the abseess was preceded for some time by pain in the abdomen, which usually occurred two or three hours after meals. In one ease it was so intense and continuous that it was regarded as a neuralgia of the cœliae plexus, while in another it seemed to radiate from the navel to the right hip. Vomiting was present in nearly half of the eases, and usually afforded relief to the pain; but in one instance it appears to have been dependent upon hypersecretion, and the stomach was found to be greatly dilated after death.

As a rule there was a history either of hæmatemesis or melæna, and some had suffered from several attacks of hæmorrhage. Loss of flesh and disinclination for food were prominent symptoms in all, and anæmia was also a noticeable feature in most of them.

The onset of perforation was always marked by violent pain and eollapse, and with a few exceptions the disease terminated fatally within a fortnight. The usual eause of death was exhaustion and sapræmia, but four died from peritonitis, three from hæmorrhage, two from secondary inflammation of the thoraeic organs, and one from pyæmia, with abscess of the parotid gland.

Physical Signs.—The signs which denote the presence of a perigastrie abseess vary according to the position of the sac and the nature of its contents. In the early stages of the complaint physical signs are often absent, and we can only surmise its existence from the history and general symptoms of the case. When the abseess is situated between the liver and the diaphragm, as in the acute form of the disease, the left side of the epigastrium and left hypochondrium are distended, and extremely tender on palpation. The abdominal muscles are rigidly contracted, and no movement of the

diaphragm can be detected upon inspiration. As the pus accumulates, the fulness of the upper part of the abdomen becomes more noticeable, and the left wing of the diaphragm is pushed upwards. Occasionally this muscle recovers its contractile power to some extent, so that on deep inspiration a shallow depression may be observed to cross the intercostal spaces on the left side ('diaphragm phenomenon'). At the same time the affected side of the chest becomes apparently enlarged, and its lower ribs are thrown outwards, so that the costal angle is increased. The intercostal spaces are also widened, and sometimes bulge slightly. The displacement of the liver and spleen is obscured to a great extent by the rigidity of the abdominal wall, but careful percussion will sometimes show that the left lobe of the liver projects into the epigastrium. In almost every instance the heart is tilted upwards, and its apex may be felt in the fourth costal interspace rather to the left of its normal position. The base of the left lung is compressed and partially deprived of air, so that the percussion note over the left posterior base is comparatively dull, and the respiratory sounds are diminished. This condition is distinguished from pleuritic effusion by an increase of tactile fremitus and vocal resonance, and by the occasional existence of moist crepitations. The presence of gas beneath the diaphragm gives rise to a tympanitic note over the front of the left chest, the upper border of which may extend as far as the fourth rib or even higher. This resonant area may also be traced across the sternum to the right nipple line, and downwards to the right of the ensiform cartilage, where it merges with the hyper-resonance over the epigastrium and left hypochondrium. The latter sometimes gives place to dulness as the pus encroaches over the cardiac end of the stomach, or the omentum becomes thickened by inflammatory exudation.

On auscultation over the front and lateral aspect of the left chest the vesicular marmar is either absent or is replaced by loud amphoric breathing. This latter phenomenon is usually due to an alteration of the breath sounds by transmission through the gas-containing cavity, but it is also possible that in some cases a communication between the stomach and the abscess permits the interchange of gas with each movement of the diaphragm. When the subplirenic

cavity contains a quantity of both pus and gas, metallic tinkling can often be heard after coughing, and a loud succussion sound may be produced by movements of the body. Finally it may be noticed that the *bruit d'airain* may be heard over the tympanitic area when coins are clinked together, while the heart-sounds occasionally acquire a metallic character.

An abscess situated between the right lobe of the liver and the diaphragm seldom contains much gas, so that its physical signs are somewhat different from those just described. The elevation of the diaphragm displaces the apex of the heart upwards and to the left, and thrusts the right lobe of the liver downwards, so that its lower edge may be felt several inches below the costal margin. The compression of the right lung gives rise to comparative dulness at the base of the chest before and behind, with diminished respiration or moist râles. Above the dull area the percussion note is hyper-resonant and the expiration prolonged. When, however, the pus is largely mixed with gas, a band of hyper-resonance intervenes between the compressed lung and the upper border of the liver, and the other indications of subphrenic pyo-pneumothorax may be detected. These various signs are most distinct within the first week, since after that time they often become modified by exudation into the pleural cavity or by inflammation of the lung. Pericardial and pleuro-pericardial friction sounds are not infrequent at this stage of the complaint.

The formation of a circumscribed tumour is a rare result of perigastric suppuration, and is chiefly met with in the chronic variety of the disease. When it occurs it is usually situated in the left hypochondrium, or in the epigastrium slightly to the left of the median line. At first the outlines of the swelling are indistinct, but as it approaches the surface they become more defined. The percussion note varies: in some cases it remains dull throughout, in others it is at first dull and afterwards resonant, while occasionally the note varies with the position of the body, showing conclusively that the abscess contains both gas and pus. In almost every instance the tumour is very tender, and deep inspiration gives rise to pain. As a rule it does not move on respiration, nor can it be displaced by pressure with the hand, owing to adhesions. When the abscess points externally, the subcutaneous tissue becomes cedematous and the skin red, while, if its contents are discharged

into the chest, it suddenly disappears. In those rare cases where the pus finds its way into the stomach or intestine, the tumour suddenly lessens in size and its percussion note becomes

tympanitic.

When the abscess is situated behind the stomach in the lesser cavity of the peritoneum, no physical signs whatever may be discovered, and hectic fever, with increasing debility, may be the only symptom of the disease (case 52). In a few cases a swelling or sense of resistance is stated to have been detected in the umbilical region, which was tender on palpation, and resonant on light, but dull on deep percussion. Occasionally pus and shreds of necrotic tissue are found in the vomit. If the pus eventually reaches the upper surface of the liver, the ordinary signs of subphrenic abscess are manifested.

The physical signs of periduodenal abscess are similar to those already described. In more than 50 per cent. of the cases a localised abscess forms a tumour in the right hypochondrium or umbilical region, while in about 30 per cent. the pus burrows behind the peritoneum in the direction of the right iliac fossa. This interesting condition is illustrated by the

following cases:-

Case LIX. A man, aged 19, was admitted into the London Hospital on November 1, 1881. In the afternoon he had fallen upon his abdomen from a height of twelve feet; he was picked up by his fellow-workmen and earried home. He vomited everything he took, and was brought to the hospital the same evening. Very little history of his previous state of health could be obtained, but it was stated by his friends that he had been drinking heavily for some time, and that vomiting had commenced two days before the accident.

On admission, the face was pinched and anxious, the pulse very feeble, the skin hot and bathed with perspiration, and he complained of slight pain in the hypogastrie region; temperature 100.4°.

Nov. 3.—The pain was localised in the right inguinal region; howels confined.

7th.—He was very restless, and had fixed pain in the right inguinal region; diarrhoa for two days.

15th.—He seemed to be in a typhoid condition; fulness was noticed in the right inguinal region, and there was some dulness there on pereussion.

18th. There was a distinct fulness close to the pubes in the right inguinal region, which was hot and tender; the stools were formed. but offensive.

He gradually improved after this date, and left the hospital on December 24. His temperature varied between 100° and 103° until November 21, after which it rose every evening to 102°, falling in the morning to the normal point.

He was re-admitted on February 1, 1882. He was then somewhat emaciated, his cheeks thin and flushed, and his expression anxious. There was a hard swelling in the right groin above and to the right of the centre of Poupart's ligament, which fluetuated with an

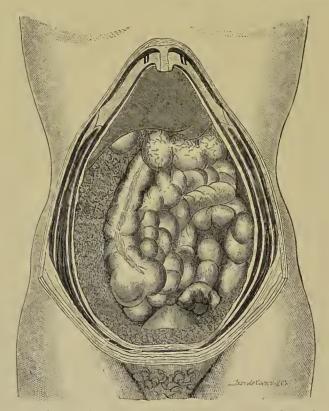


Fig. 53.—Sketch of a case of periduodenal abscess, where the pus pointed in both groins.

emphysematous erackling, as if air and fluid were intermixed; bowels opened daily. He had little pain or tenderness over the abdomen.

10th.—An incision was made into the tumour, and a large quantity of yellowish feculent pus was evacuated.

27th.—The discharge was feeulent and profuse, and pressure over any part of the abdomen caused a large quantity to be evacuated.

June 14.—The sinus was more freely opened, and a large amount of fæeal matter removed.

28th.—Diarrhœa eame on, and he became much weaker.

He lingered until July 31, when he sank from exhaustion, nine months after the accident. The temperature always rose in the

PERIGASTRIC AND PERIDUODENAL ABSCESS 339

evening to 101° or 102°, and generally fell to the normal point in the morning.

Autopsy.—The body was so much wasted that it looked as if the patient had died of starvation. On opening the abdomen a channel was discovered extending from the under surface of the liver to Poupart's ligament on the right side; it was situated behind the ascending colon, and was about two and a half inches wide. The roof and inner wall were formed of thickened peritoneum and the adhering gut, the floor by the thickened fascia covering the quadratus lumborum and the ileo-psoas, the outer wall by the thickened transversalis fascia, and there was a free opening at Poupart's ligament, where a fæcalstained cavity was situated. The lower horizontal limb of the channel ran behind the pubes, and terminated in a slightly sacculated cavity in the left groin. The channel contained no solid fæces, but its walls were coated with a brownish-yellow fæeal deposit. As it was supposed there might be some connection with the intestines, water was injected into the colon and into the small intestines, but none escaped until it reached the duodenum, from which part it flowed out in a large stream into the upper extremity of the channel, and swept downwards to Poupart's ligament. At the point of escape the duodenum presented an ulceration the size of a sixpence, with thickened edges, about five inches and a half from the pylorus, which had perforated all the coats and communicated with the channel. All the other organs of the body were healthy (fig. 53).

Case LX. A man, aged 32, was admitted into Guy's Hospital under the care of Dr. Goodhart for painful swelling of the abdomen, with pyrexia. Five weeks before, he had been seized with sudden severe abdominal pain and vomiting, three days after which he had an attack of pleurisy on the right side. On admission the abdominal wall was stretched and shiny, and there was marked fulness in the flanks. No ascites could be made out. When he had been in the hospital for four days an ineision was made in the right iliac region, and eight ounces of pus evacuated. Ten days later a second abscess was opened above the left groin. Eventually two more incisions were made, one in each loin, and from all four openings pus continued to drain until his death, six months after the onset of his illness.

Autopsy.—The abdominal viscera were found to be firmly matted together by old adhesions, and there were several collections of pus behind the peritoneum. In the duodenum, immediately below the pylorus, there was upon the posterior wall a thick-edged perforating ulcer, half an inch in diameter (recored by Perry and Shaw).

In the next case the patient recovered after presenting symptoms and physical signs very similar in character to those just mentioned.

Case LXI. A gentleman, 27 years of age, after suffering for some months from pain and 'dyspepsia,' was suddenly attacked on May 2, 1894, with violent pain in the abdomen, and eollapse. The next day there were great pain and tenderness in the right hypochondrium and to the right of the navel, and the wall of the abdomen was rigidly contracted. The pulse was 84, and the temperature 100.6° .

May 7.—The pain is more or less constant over the right side of the abdomen, and is increased by lying upon the left side. The breathing is quick and shallow, and there has been some sickness. The bowels are confined. The abdomen is retracted, and very tender over the course of the ascending colon, but no tumour can be felt. The lungs are clear, and the urine is normal.

May 9.—The patient was suddenly seized this morning with acute pain in the right lumbar region, just below the kidney, where there is great tenderness. No blood in the urine. Temperature 101° F.

May 13.—The pain in the right side of the abdomen and back persists, and he now complains of pain in the left groin, which is somewhat tender upon pressure and presents slight increased resist-

anee on palpation.

May 15.—There is an indefinite swelling to be felt in the left groin just above Poupart's ligament, with comparative dulness. The pain in the right loin continues, and a kind of 'squishing' sound is elicited on percussion. The temperature varies between 100° and 101.5°. The pain is increased after an action of the bowels.

May 29.—The patient has continued in much the same condition since the last note. The swelling in the left groin has not developed, and there are no fresh physical signs. The temperature

is elevated at night, but falls to normal in the morning.

May 30.—Yesterday evening patient had an attack of diarrhœa and passed a large quantity of pus, after which he became somewhat collapsed. The pain in the abdomen has disappeared, and the percussion note in the loin is now clear.

Subsequently he began to improve, but convalescence was retarded by swelling and cedema of the right leg. When this subsided the progress towards recovery was rapid. The temperature never rose above the normal, the pain did not return, and the patient

steadily gained in weight.

When we saw him on Sept. 25th, 1897, the following note was made: There is now no pain in the abdomen, but the patient is subject to flatulence and discomfort after food, and to constipation. When he lies upon his back there is a well-marked hollow in the left hypochondrium, with a corresponding swelling on the right side. The tumour is clastic, resonant on percussion, affords a splashing

sound on palpation, and is not altered by the position of the body. The stomach cannot be detected in its usual position. There is comparative dulness over the right lumbar region in front and behind, and the loin appears to be somewhat fuller than on the opposite side. In the erect posture the deformity of the abdomen is still more conspicuous, and peristaltic movements can be seen over the swelling in the right hypochondrium.

Although the notes of the above case are very scanty, there can be little doubt that there was originally perforation of a duodenal ulcer with suppuration in the retroperitoneal tissue. It is also probable that the pus found its way behind the ascending colon, as in case 59, and also into the perinephritic tissue, but that instead of pointing in the iliac fossa it fortunately burst into the colon and was evacuated by the bowel. The case is also of considerable interest as showing the deformity of the abdomen which may arise from contraction of the abscess cavity.

These facts appear to indicate that the symptoms and signs of retroperitoneal abscess from perforation of a duodenal ulcer are somewhat as follows: After the initial symptoms of perforation have passed away there is constant pain in the right side of the abdomen, accompanied by an elevation of temperature, and great tenderness over the hypochondrium and to the right of the navel. If the pus makes its way behind the colon in the direction of the iliac fossa, the area of tenderness increases, and a swelling appears above Poupart's ligament on the right side which presents the ordinary features of an abscess. If unrelieved, the pus may travel across the hypogastrium and present in the other groin, or it may gravitate into the pelvis and point beside the rectum. Occasionally the perinephritic tissue is invaded, and great pain is experienced in the loin, with swelling, tenderness, and perhaps a 'squishing' sound on percussion owing to the mixture of gas and pus. In these cases the matter may either present in the loin or burst into the colon.

Complications.—Secondary abscesses are an occasional result of subphrenic suppuration, and in the absence of a reliable history of gastric perforation their presence is apt to prove very misleading. The difficulty of diagnosis is well shown in the following case, where, in addition to three collections of pus in the abdomen, there was also an empyema of the left side.

Case LXII. A man, aged 47, was admitted into the London Hospital for a swelling in the abdomen. The history he gave was extremely vague, but it appeared that for eighteen months he had suffered from pain after meals, and that, one month before his admission, he had been attacked by severe pain in the region of the stomach. On examination a mass the size of a cricket-ball was discovered in the left iliae fossa, which was nodular and painful on pressure; dulness on percussion and distant breath-sounds were also found at the base of the left chest. A few days afterwards the abseess was opened and eight ounces of very fetid pus were evacuated. This was, however, followed by erysipelas, and as his breathing became difficult the left chest was aspirated and forty ounces of pus removed.

Post-mortem examination.—It is stated that there 'was contraction of the descending colon and an abseess in the abdominal walls,' but the notes on this point are very scanty. 'There was also an irregular contraction of the stomach around a point midway between the cardiac and pyloric ends, and at the base of this a circular depression which was adherent to the left lobe of the liver. External to the adhesion there was a small abscess below the stomach, left lobe of liver, and diaphragm. The stomach was much thickened around the ulcer, and an abscess the size of a hen's egg was situated below the eapsule of the spleen. The left pleura contained pus.'

In the next case the obscurity of the initial symptoms and the discovery of an inflammatory tumour in the pelvis seem to have led at first to a diagnosis of pelvic peritonitis.

Case LXIII. A single woman, aged 18 years, was admitted into the Royal Hospital, Sheffield, on March 30, 1898, under the care of Dr. Hall. The menses began when she was fifteen, but since that time she had seen nothing. When thirteen she had inflammation of the bladder and pain on micturition. For the last six months she had had pain on micturition. For three years she had suffered from indigestion, with pain and vomiting after food, for which she had been treated for the last few months. Both her parents died of consumption. About the seeond week in March she fell downstairs and hurt her left side badly, so that she was forced to go to bed for five days. After this she got up and tried to work, but was unable to do much. On March 25 she had acute pain in the left side, and took to bed. On March 28 she complained of difficulty of breathing, vomiting, and diarrheea.

On March 29 her condition was as follows: Anxious expression, bright flush on both eheeks. Tongue moist, coated down the centre. Respirations 34. Pulse 120; regular, small volume. Temperature 101·2°. Abdomen much distended all over, tender and tympanitic. Diarrhæa, motions not light-coloured. Small swelling felt in left iliac

region about the size of an orange, indistinct and tender. Per rectum: ill-defined tender swelling in front and to the left of bowel. Chest: cardiac impulse fourth space behind nipple. Respirations entirely thoracic; the diaphragm did not move at all, so far as could be seen. Impaired movement of left base with well-marked dulness behind for about two fingers' breadth. Diminished entry of air into left side of ehest. Tympanitic resonance right up to eardiac impulse in front and in axilla. Friction-sounds over left base in front. Some rhonehi in right lung. Frequent cough, often very harassing; slight mucous sputa. Urine 1024; faint cloud of albumen. Belladonna fomentations and morphia internally were prescribed, which relieved the pain and distension considerably. The temperature and other symptoms continued much the same, the cough troubled her a good deal, but the abdominal pain and swelling became considerably less. On April 8, about 8.30 A.M., she was seized with severe dyspnœa, and began to expectorate large quantities of gruel-like, highly offensive, purulent matter. The distress of breathing became very great, and she gradually became eyanosed and died of respiratory failure about three hours later.

Autopsy.—A large subphrenie abscess was found below the left wing of the diaphragm, due to the perforation of a chronic ulcer near the centre of the lesser curvature. The left lung was firmly adherent to the diaphragm, which had been perforated during life and had allowed some of the pus to escape into a bronchial tube. The pelvis contained a large abscess on the left side, the walls of which were formed by matted intestine, and by adhesions between the uterus, intestine, and broad ligament.

Occasionally pylephlebitis takes place with the formation of secondary abscesses in the liver. The symptoms of this complaint are often masked by those of the primary disease, but as a rule severe pain is complained of in the right hypochondrium, followed by jaundice. The liver rapidly increases in size, repeated rigors occur, and death ensues within a few days. In some instances pyæmic abscesses develop in the lungs, or a murmur becomes audible over the mitral area of the heart owing to acute endocarditis.

The rupture of the abscess into the pleural cavity is usually accompanied by sudden pain in the chest, extreme dyspmæa, and collapse. In one of our cases the accident was ushered in by repeated rigors, while in two others the patients felt that something had given way in the chest. In one instance death ensued within a few hours from shock.

The physical signs of this condition vary considerably. If

the subphrenic abscess contains much gas, the initial phenomena will be those of pneumothorax; but in most cases the gas becomes rapidly absorbed, and the accident is followed by the signs of empyema. When the lung is adherent to the diaphragm and the abscess finds its way directly into a bronchial tube, the patient experiences a sudden pain and expectorates a large quantity of pus (case 63). If life is prolonged, coagulated milk and other particles of food may appear in the sputa.

In all cases where the abscess bursts into the pericardium the initial symptoms are sudden pain over the præcordium and urgent dyspnæa. The pulse is very small and rapid, the extremities become cold, and the patient exhibits great restlessness. If, as is usually the case, the perforation is very small, acute pericarditis is set up, and a loud friction sound may be heard over the region of the heart. If, however, a large quantity of gas finds its way into the sac, the præcordial dulness is replaced by hyper-resonance, and the heart-sounds either become inaudible or acquire a metallic character; while in those cases where the pericardium is filled with pus, the ordinary signs of pericardial effusion manifest themselves. As a rule the patient dies from cardiac failure within four days of the accident.

In those rare instances where the abscess bursts into the colon there is a sudden access of pain in the abdomen, followed by the evacuation of pus, a fall of temperature, and relief of the pain. In the following case hæmorrhage seems to have ensued.

Case LXIV. A woman, aged 48, was seized, whilst eating her luncheon on May 2, with a sudden violent pain in the epigastrium, cramp-like in character, which lasted with intense severity for half an hour, during which period she felt sick but did not actually vomit. Previous to this attack she had always enjoyed good health, and had never been troubled with pain or sickness after food. She was put to bed, and on the 8th or 10th of the month diarrhea set in. During this time, although she occasionally vomited, she was able to take her food without any increase of the pain. The diarrhea continued until her admission into the hospital on June 2. She was then very thin and anæmic. She lay upon her back or left side, as it was painful to recline upon the right side. The temperature was almost normal. The liver was much enlarged, and near the umbilicus an indefinite tumour could be detected. On June 3 she passed about six ounces of blood with the stool, and on each of the

two succeeding days voided about the same amount, after which she

Autopsy.—An abscess was found situated between the enlarged liver, the anterior abdominal wall, and the coils of intestine. The abscess communicated with the transverse colon and the duodenum. The aperture in the colon was close to the hepatic flexure, was only large enough to admit a small probe, and was apparently of recent origin. That in the duodenum was upon the posterior wall, was circular in form, as large as a sixpence, and had smooth edges. At the middle of the lesser curvature of the stomach was a large chronic ulcer whose base was adherent to the liver.

Dr. West, who records the case, remarks that the sudden illness of the patient is probably to be explained by the rupture of the duodenal ulcer and the consequent formation of the abscess. The abscess eventually burst into the colon, and its rupture probably explains the occurrence of blood in the stools during the last few days of life.

Rupture of the abscess into the *peritoneal cavity* is followed by collapse and general peritonitis (case 53), while in those exceptional cases in which the pus perforates the stomach the accident is portrayed by the vomiting of large quantities of pus.

Diagnosis.—When a patient who has suffered for some time from the symptoms of gastric ulcer is suddenly attacked by pain in the epigastrium and collapse, followed by tenderness over the upper part of the abdomen and fever, perforation of the stomach with localised peritonitis must be suspected. This becomes a certainty if, in addition to these signs, there is distension in the left hypochondrium, hyper-resonance or a bell-note over the front of the chest as far as the fourth rib, with tilting up of the apex of the heart, paralysis of the diaphragm, and signs of compression of the base of the lung. When, however, as in case 52, there is no history of a previous gastric disorder, and a total absence of pain and physical signs in the abdomen, the diagnosis of perigastric abscess may be impossible.

In the early stages of the complaint secondary inflammation of the thoracic organs is very apt to divert attention from the primary disease, and several instances have been recorded in which the symptoms were erroneously ascribed to the presence of the pleurisy, pneumonia, or pericarditis. In all such cases, however, it may be remarked that the patient appears to be more profoundly ill than the physical signs seem to warrant, and that he often exhibits the pinched and septic appearance

which is so characteristic of peritonitis. It is also noteworthy that the signs of the thoracic complication differ in several respects from those which accompany the usual forms of inflammation of the pleura or lung. Thus in every instance the percussion note over the front of the chest is tympanitic, the apex of the heart is pushed upwards or towards the affected side, while the signs of pleurisy or pneumonia are often confined to the back of the lung. It is also to be observed that there is marked rigidity of the abdomen, with some degree of swelling of the epigastrium and left hypochondrium, and great tenderness upon pressure.

The diagnosis of subphrenic suppuration from empyema can usually be made by attention to the following points: In the former complaint the heart is pushed upwards, there is resonance in front and dulness behind, the bruit d'airain can often be obtained, the respiratory sounds are audible over the back of the chest, and tactile fremitus may be increased. In empyema, on the other hand, there is dulness on percussion both before and behind, the apex of the heart is displaced to the opposite side, and the breath sounds, vocal resonance, and tactile fremitus are absent over the dull area.

When the subphrenic abscess is accompanied by effusion into the pleura, the diagnosis is rendered much more difficult, especially if the introduction of a needle has shown the existence of pus. In this condition, however, it may be observed that the outflow of the pus is accelerated by inspiration owing to the compression of the sac by the downward movement of the diaphragm, while the reverse is the case in ordinary empyema. In some of the recorded cases, the surgeon was surprised to discover clear fluid in the chest when he expected pus, and this led to a more careful examination of the case; while in others the discovery of clear fluid above and pus at a lower level indicated at once the existence of two effusions separated from one another by the diaphragm.

In a few instances the disease has been mistaken for simple pericarditis, owing to the existence of a pericardial friction sound. As a rule, however, the tympanitic resonance over the front of the chest, with deficient movement of the base, and the swelling and tenderness of the epigastrium, are sufficient to indicate the coexistence of subphrenic mischief.

When the cavity is large and contains both gas and pus.

it is very apt to be confused with pyo-pneumothorax. In the last-named disease, however, there is usually a history of cough, dyspnæa, hæmoptysis, and of other symptoms indicative of pulmonary disease; whilst in perigastric abscess the patient has previously complained of pain after food, or dyspepsia. In pyo-pneumothorax, the whole of the affected side is enlarged,

the intercostal spaces are widened, and the movement of the ribs is annulled. In subphrenic abscess, only the lower part of the chest is distended and motionless, while the upper portion moves with respiration. In pyo-pneumothorax the heart is pushed towards the opposite side; there is hyper-resonance over the upper part of the chest, with dulness at the base, which varies with the position of the body; while the respiratory sounds are either absent altogether, or amphoric breathing can be heard over the resonant area. In subphrenic abscess the heart is pushed upwards; there is tym- 1. Whole side of chest distended and immobile. 2. Heart displaced to opposite side. upwards; there is tympanitic resonance over the
front of the chest as far as the third or fourth rib,

1. Whole sale of chest distended and minioble.
2. Heart displaced to opposite side.
3. Upper part of chest hyper-resonant, with bruit d'airain; lower part dull, but varying with position of patient.
4. Breath-sounds absent, or amphorie over upper part; tactile fremitus and vocal resonance abolished.
5. Succussion and metallic tinkling limited to chest. and posteriorly the note is

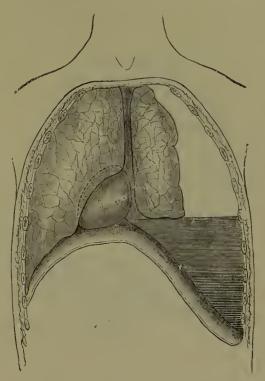


Fig. 54.—Diagram of a case of pyopneumothorax.

Physical Signs.

dull in any position the patient may assume; the breath-sounds are normal over the upper part of the chest, deficient or slightly tubular over the posterior base, while anteriorly amphoric breathing can often be heard as far down as the level of the umbilicus. In both diseases the bruit d'airain, the succussion sound and metallic tinkling may be obtained, but in perigastric abscess they are audible over the upper part of the abdomen as

well as over the chest. The appended diagrams (figs. 54, 55) serve to emphasise these distinctions.

When perforation of an ulcer near the pylorus gives rise to gradual extravasation of the gastric contents, the acute pain in the epigastrium, with vomiting and fever, is apt to be mistaken

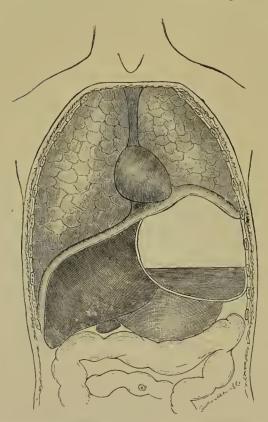


Fig. 55.—Diagram of a case of perigastric abscess containing gas and pus.

Physical Signs.

1. Lower part of chest distended and immobile.

hower part of chest distinct and immoore.
 Apex of heart tilted up.
 Hyper-resonance from fourth rib downwards, with bruit d'airain; comparative dulness at base behind.
 Breath-sounds normal over upper part of chest; amphorie below fourth rib and in hypochondrium;

deficient behind. 5. Succession and metallic tinkling over upper part of abdomen.

for biliary colic. latter affection, however, is far more common in women than in men, while the converse is observed in the gastric complaint. The fever in subphrenic abscess continues after the immediate symptoms have passed away, the pain is constant, jaundice is usually absent, and the signs of thoracic complications soon make their appearance. It must be remembered, however, that suppuration around the gall-bladder is not an uncommon cause of subphrenic abscess, but in such cases the sac contains no gas. Osler has recorded an instance of perinephritic abscess which perforated the colon, and gave rise to a gas-containing cavity beneath the diaphragm.

When a perigastric abscess devoid of gas has

formed a palpable tumour, it may be mistaken for a hydatid cyst in a state of suppuration. But the history of a hydatid cyst is very characteristic: it is often discovered by accident, and has generally been unattended for a considerable length of time by pain or any other inconvenience, excepting such as may arise from its bulk; there has been no previous emaciation or

deterioration of the general health, and neither pain after food nor vomiting. A hydatid tumour is always dull on percussion, is not lessened by diarrhoa or vomiting, can be generally shown to be connected with the liver, and, unless adhesions have been extensively formed, it moves with the respiration and upon pressure.

A perigastric abscess may also be mistaken for an hepatic abscess, unless it is borne in mind that the latter almost always arises from dysentery or an affection of one of the abdominal organs whose circulation is connected with the portal system. It chiefly occurs in persons who have lived in the tropics; its course is acute, and is usually attended with repeated rigors; the tumour is quite dull on percussion, and is never accompanied by signs of pneumothorax.

Treatment.—As soon as the existence of the abscess can be determined the cavity should be opened and drained; for every day that the operation is postponed increases the risk of secondary inflammation of the thoracic viscera and perforation of the diaphragm. The abscess is most conveniently opened in the left hypochondrium, care being taken not to disturb the adhesions between the stomach and the abdominal wall, which prevent extravasation into the general cavity of the peritoneum. When the pus is situated in contact with the diaphragm a counter opening may be made between the ribs behind in order to secure free drainage.

LITERATURE

Allbutt, Clinical Journal, June 7, 1893.

Barlow, London Medical Gazettc, 1845, p. 13.

Broadbent, Brit. Med. Journ., Oet. 30, 1897.

Cossy, Archives Générales de Médecine, 1879, tom. 2.

Coupland, Brit. Med. Journ., 1889, vol. i. p. 636.

Debove & Rémond, Gazette des Hôpitaux, 1890, p. 1159.

Fagge, Guy's Hospital Reports, 1874, third series, vol xix. p. 213.

Fenwick, Clinical Lectures on Obscure Diseases of the Abdomen, 1889, p. 119.

Fitz, Boston Med. and Surg. Journal, Dec. 18, 1890.

Hall, Clinical Journal, 1898, p. 354.

Leith, Edinburgh Hospital Reports, 1895, vol. iii.; Internal Clinies, 1895, vol. iv. p. 49.

Lenoir, Bull. Soc. Anat., Nov. 11, 1890.

Leyden, Zeitsch. f. klin. Med., 1880, Bd. 1.

Leyden & Renvers, Berl. klin. Woehen., 1892, No. 46.

Lierman, Deut, med. Woehen., Feb. 2, 1892.

Mason, Trans. Assoc. Amer. Phys., 1893, vol. viii. p. 218.

Maydl, Ueber subphrenisehe Abscesse, Wicn 1894.

Nowak, Schmidt's Jahrb. 1891, Bd. 232, p. 73.

Penrose & Dickinson, Clin. Soc. Trans., vol. xxvi. p. 72.

Perry & Shaw, Guy's Hospital Reports, 1893, vol. i. p. 171.

Sänger, Archiv f. Heilkunde, 1878.

Scheurlen, Charité-Annalen, 1889.

Schreiber, Deut. Arch. f. klin. Med., 1883.

Scnator, Charité-Annalen, 1884.

Strecton, London Medical Gazette, vol. iii. p. 43.

West, Clin. Soc. Trans., vol. xix. p. 113.

CHAPTER VI

GASTRIC FISTULÆ

Perforation of the Pleura, Pericardium, Heart, Mediastinum, Colon, and Skin-General Emphysema-Air in the Blood-vessels

[To be read with pages 68-74]

Perforation of the Diaphragm.—Direct perforation of the diaphragm by an ulcer of the stomach is extremely rare. The total number of recorded cases which were verified by an autopsy amounts to only about twenty-nine, while Biach found that, out of the 918 examples of pyo-pneumothorax which he collected, only two were due to a gastric ulcer. The accident usually occurs in women from 35 to 50 years of age, who have suffered for a long time from pain after food or hematemesis. It may take place quite suddenly and without any warning, or it may be preceded for several days by incessant pain in the epigastrium and at the base of the chest. Occasionally the patient suffers from attacks of asthma, palpitation of the heart, or from pain and tenderness along the course of the phrenic nerves in the neck. As a rule the rupture occurs when the stomach is full of food, or during a violent effort with the glottis closed. In most instances the hole in the diaphragm does not exceed the size of a quill, but both Günsburg and Needon have recorded cases in which the opening was so extensive that the spleen and a part of the stomach found their way into the thorax.

(1) Perforation of the Pleura and Lung.—The results of perforation of the left side of the chest vary according to the condition of the pleura at the time of the accident, and the size of the aperture. If the base of the left lung is free from adhesions, the gastric contents will find their way directly into the serous sac and excite suppurative pleurisy, with or without

pneumothorax. On the other hand, if the two layers of the pleura have previously become united, the ulcer, after penetrating the diaphragm, will gradually erode the base of the lung, and produce a gangrenous eavity in its substance. Occasionally this latter condition is replaced by a pulmonary absecss which finally bursts into a bronchial tube. Perforations of the diaphragm almost invariably terminate fatally, notwithstanding the fact that Sturges and other writers have published cases where pyo-pneumothorax, which had apparently arisen in this manner, ended in spontaneous recovery. Ransom has recently recorded an instance which shows the beneficial results of surgical treatment.

Empyema.—In three out of our eleven cases of perforation of the left pleura, the patient was attacked with an acute suppurative pleurisy, while in another 'exudation' was found after death. It is probable, therefore, that in about one-third the accident is followed by the symptoms of pleural effusion. The occurrence of the perforation is marked by sudden and severe pain in the left side of the ehest, and is followed immediately by dyspnæa and collapse. After the disappearance of the shock the temperature rises, stabbing pain is experienced on deep inspiration, and a friction-sound may be detected over the affected side. The epigastrium and left hypochondrium are somewhat distended and tender, and vomiting is often a troublesome symptom. Within a short time dulness on percussion, displacement of the heart, and immobility of the lower part of the ehest, with an absence of tactile fremitus, voeal resonance, and the vesicular murmur, indicate the presence of a pleural effusion, while the extreme gravity of the general symptoms shows that the disease has arisen from an exceptional cause. Death may ensue within forty-eight hours, or life may be prolonged for a week or more.

Pneumothorax and Pyo-pneumothorax.—In four out of the eleven cases (36 per eent.) a considerable quantity of gas escaped from the stomach into the pleura, so that the first signs of the accident were those of pneumothorax. In this condition the pain and collapse which attend the act of perforation are very severe, and the patient suffers from urgent dyspnea. The left side of the chest is somewhat distended and moves but little on respiration; the heart is displaced to the opposite side, the percussion note is hyper-resonant, and the

bruit d'airain may be obtained, while the vesicular murmur, tactile fremitus, and vocal resonance are abolished. In Müller's case death ensued from cardiac failure at a comparatively early stage of the complaint; but in the other three, secondary pleurisy occurred with the formation of pus. This condition of pyo-pneumothorax is accompanied by continued fever and urgent dyspnæa on exertion. The lower part of the chest becomes dull on percussion, and metallic tinkling or a succussion sound may be detected if the fluid accumulates in any quantity. Death usually takes place within ten days.

Case LXV. A woman, 39 years of age, was admitted into the London Hospital in a state of collapse, with urgent dyspnæa. She stated that she had suffered from pain in the stomach for about four years, and had often vomited her food. On one occasion the vomit had contained a quantity of blood. There had never been any cough or other symptoms of phthisis. The present illness commenced quite suddenly the day before, with a sensation of something 'giving way 'in the chest, followed immediately by great pain below the left breast, breathlessness, and sickness. On examination she was found to be somewhat cyanosed, with a feeble and irregular pulse, and had to be propped up in bed on account of dyspnea. The left side of the chest was moderately distended and motionless, the percussion note hyperresonant, the apex of the heart displaced under the sternum, and the respiratory sounds absent. Distant amphoric breathing was audible over the anterior base of the chest and epigastrium. The patient died on the third day.

Autopsy.—The left lung was collapsed, and the pleura contained a considerable quantity of gas and thin purulent fluid, mixed with milk and undigested food. On the posterior wall of the stomach, close to the lesser curvature, and about two inches from the cardiac orifice, was a circular chronic ulcer the size of a florin, the base of which was formed entirely by the diaphragm. On careful examination a small perforation was found near the upper margin of the ulcer, through which a small catheter could be passed directly into the left pleural cavity. The other organs were healthy.

Case LXVI. 'The patient, a single woman, aged 20 years, when first seen was apparently in the last stage of phthisis, from which disease, indeed, she was supposed to be suffering. Inquiry, however, elicited the fact that for a couple of years she had suffered from pain after food, with occasional vomiting, and that some months previously she had vomited dark blood. Six weeks previously to being seen, while she was shelling peas, the patient was suddenly seized with stabbing pains in the left axilla shooting across the stomach. Since then she

had kept her bed, had had frequent vomiting of food and bile, and had coughed up phlegm of an unpleasant taste and odour to the extent, it was said, of half a pint in the 24 hours.

'Examination showed extreme emaciation and an anæmic dry skin. The abdomen was flat, not very tender, and the stomach resonance mounted high near the heart. In the chest the resonance at the apices was poor, but there were no crepitations or bronchial breathing there. There was dulness only in the hinder and lowest part of the left axilla, over the greater part of which the note was sub-tympanitic. In the lower two-thirds of the left axilla and at the left base were amphoric breathing and metallic tinkling. There was no succussion splash. The diagnosis being made of pneumothorax from perforating gastric ulcer, the patient was advised to come into hospital, but she

only did so a week later.

'On admission (September 26, 1898) the abdomen was found to be flat and did not expand well during inspiration in its left half. There was some tenderness on deep pressure in the left hypochondriac and lumbar regions. On the front of the chest there was fair resonance under both clavicles and the breath sounds were normal. The cardiac dulness reached from the fourth space up to the lower edge of the second rib; below this was a high-pitched tubular note extending outwards to just outside the nipple line and downwards for two inches. Below this there was stomach resonance. Outside the nipple line and in the axilla, below the nipple level, was an area of absolute dulness which was continued posteriorly at the same level to within two inches of the spine. Over the greater part of this dull area both breath and voice sounds were absent; only at its upper margin were there distant bronchial breathing and ægophony. It was thus evident that a large amount of fluid had accumulated in the chest during the seven days' interval between the two examinations. A needle inserted near the angle of the left scapula withdrew some fætid pus. On September 29 the patient was anæsthetised with A.C.E. mixture and two inches of the seventh left rib were excised near the angle of the scapula. Only a little gas escaped at first, the lung being slightly adherent to the chest wall, but on the finger being introduced in an upward and forward direction about four ounces of fætid pus escaped. The cavity was plugged with iodoform gauze. Hypodermic injections of morphia were ordered to check the cough and relieve the pain, and food was given solely by enemata. Some hours after the operation considerable dyspnæa set in and the patient coughed up some bloodstained mucus. The same evening the temperature rose to 103.8° F., having in the three previous days not exceeded 100°. On September 30 the patient was rather better, though she was coughing up besides muco-pus some gastric juice and bile, and numerous coarse crepitations were audible all over the front of the left lung. The

gauze plugs had to be frequently renewed, being soaked with discharge which was, however, much less offensive.

'By October 2 further improvement had occurred, the pus of the discharge being replaced by a yellowish inoffensive fluid presenting the characteristics of gastric juice. The coughing up of stomach contents was now prevented by propping the patient in the upright position, so that the hole in the stomach should be above the level of the fluid therein. On this day some of Carnrick's liquid peptonoids and also salicylate of bismuth were given by the mouth. On October 9 the note says: "Further improvement, temperature keeps lower; no gastric contents seen in the phlegm or in the discharge from the sinus; patient has vomited once since the last note. The discharge is much less offensive and is diminishing. The sinus has been treated with iodoform emulsion. The apex of the left lung is clearing up, but a few crepitations are heard at the right base."

'On October 11 the left front of the chest showed no dulness and normal breath sounds, and on the left back resonance and breath sounds were much improved and moist sounds were fewer. The patient's general condition now, however, began to change for the worse. The temperature that night rose to 101°, vomiting recommenced, and the signs of pneumonia at the right base became more marked. Three days later, dulness, bronchial breathing, and coarse crepitations had spread over the lower half of the right lung, and on the 18th she died. The improved condition of the left lung remained, however, to the end.

'Autopsy.—On opening the abdomen the stomach was found to be distended and adherent to the liver, diaphragm, and spleen. When the chest was opened the left side of the diaphragm with the stomach adherent was seen to mount as high as the third intercostal space. The left lung was attached by firm adhesions to the chest wall, and the sinus was found to lead into a healthy granulating cavity. The left lung, though somewhat contracted, was free from pneumonia, suppuration, or tubercle, and was obviously in a fair way to recovery. There was no visible opening from the diaphragm to the stomach. The right pleural eavity, on the other hand, contained a little semipurulent fluid, and the lower half of the right lung was covered with lymph and infiltrated with grey pncumonia. When the stomach was opened an indurated ulcer was found on the hinder wall near the cardiac end, firmly adherent to the diaphragm. In the middle of this was a small perforation closed by recent lymph. The splcen was adherent to the stomach, and its inner surface was covered with a layer of pus. The upper surface of the liver was also bathed in thick green pus which extended to the right side and appeared to be infiltrating the right half of the diaphragm.' (Recorded by Dr. Ransom.)

Invasion of the Lung.—In four instances the base of the left lung was found to be adherent to the diaphragm and to have been directly invaded by the ulcer. In such cases the initial symptoms of perforation are often absent, and the patient apparently suffers from an insidious form of pneumonia. As a rule the temperature is markedly irregular, the pain and cough troublesome, and after a few days the expectoration becomes considerable in quantity, foul-smelling, and may contain traces of blood. If the gangrenous cavity communicates with a bronchial tube, curdled milk or other food may be expectorated, and sudden death may occur from suffocation owing to the contents of the stomach gaining an entrance to the lung. The physical signs are dulness on percussion over the base of the chest, tubular or amphoric breathing, with moist crepitations, increased vocal resonance and tactile fremitus. Death usually takes place from exhaustion within a fortnight.

Diagnosis.—The diagnosis of these rare complications depends upon the establishment of three facts: (1) the long-continued existence of the symptoms of gastric ulcer; (2) the sudden onset of severe pain in the chest, followed by dyspnæa and the signs of pneumothorax or pleurisy; (3) the absence of primary disease of the lung and of sub-diaphragmatic abscess.

When empyema is the only result of the accident, the diagnosis of perforation of the diaphragm is exceptionally difficult, owing to the fact that pleuritic effusion is very apt to ensue from any source of irritation beneath that structure. In every suspicious case the exudation should be carefully examined for traces of food and hydrochloric acid, and the vomit for pus. is interesting to note that in two of the recorded cases round worms were discovered in the pleural exudation. gangrene or abscess occurs at the base of the left lung, the expectoration often contains curdled milk or particles of food. Phthisis and chronic ulccr of the stomach frequently coexist in the same individual, so that in many instances of pneumothorax from the rupture of a tubercular vomica there is a history of pain after food and hæmatemesis. It is to be observed, however, that the two complaints are seldom active at the same time, and that if the symptoms and signs of phthisis are predominant the pncumothorax is almost certain to have arisen from the pulmonary disease.

Treatment. - In each case the pulmonary complication

must be treated upon the usual lines. If pus is present the chest must be drained, and if the patient appears able to bear the operation an attempt may subsequently be made to deal with the gastric ulcer. In pneumothorax an operation is seldom feasible; but if pus coexists with air the pleura should be drained as in Case 66.

(2) Perforation of the Pericardium.—Five cases have been recorded in which an ulcer of the stomach established a direct communication with the pericardium. In two instances the contents of the sac were found after death to consist principally of gas (pneumo-pericardium), while in the others both fluid and gas were present. The majority of the patients were women, the youngest being only 26 years of age. In every instance symptoms of gastric ulceration had existed for a considerable time. In two the act of perforation was accompanied by severe pain and precordial distress, but in the others dyspnœa and palpitation appear to have constituted the chief causes of complaint. Subsequently the breathlessness became urgent, the face and extremities were cyanosed, and the heart rapidly failed. The chief physical signs are due to the presence of gas in the pericardium. On examination the apex beat of the heart cannot be located, and the precordial dulness is replaced by tympanitic resonance. Occasionally a pericardial friction can be heard in the recumbent position, and as a rule the sounds of the heart acquire a loud metallic timbre. In Säxinger's case a loud metallic sound, synchronous with the pulse, could be heard at a distance of several feet from the patient. Death usually takes place within three days from cardiac failure, but in one instance it occurred within ten minutes (Fenwick), and in another the patient lived for twelve days (Säxinger).

Case LXVII. A man, 44 years of age, was admitted into hospital for dyspnæa and palpitation of the heart. His history showed that for seven years he had suffered from the symptoms of gastric ulcer. A loud metallic sound, synchronous with the radial pulse, but not influenced by respiration, was audible to a person standing in front of the patient, and when the cardiac region was auscultated the sound was so loud as to obscure the inspiratory murmur. The apex beat could not be located, and the cardiac dulness was replaced by hyperresonance. There was comparative dulness on percussion over the base of the right chest, and a pleuritic friction sound was heard over

the scapula. The patient had one or two rigors, and succumbed to ædema of the lungs on the twelfth day after the signs of the pneumopericardium had appeared.

Autopsy.—There was serous exudation in the right pleura. Close to the cardiac end of the stomach, on the lesser curvature, was a chronic ulcer which had perforated the diaphragm and established a communication between the stomach and pericardium. The pericardium was distended with gas. (Recorded by Säxinger.)

Case LXVIII. A woman, 26 years of age, was admitted into hospital for indigestion. She was much wasted and very cachectic, and presented the appearance of a person suffering from cancer of the stomach. Three weeks later the first sound of the heart suddenly acquired a loud metallic character, the cardiac dulness was replaced by hyper-resonance, the pulse was small and feeble, and the patient experienced some palpitation and dyspnæa. There was no pain. She died in about forty-eight hours.

Autopsy.—The fundus of the stomach was firmly adherent to the diaphragm, and a chronic ulcer upon its posterior wall was found to have penetrated into the pericardial sac, the aperture of communication measuring 25 cms. in circumference. The pericardium was full of gas, and the surface of the heart was covered with lymph. (Recorded by Moizard.)

(3) Perforation of the Heart.—Five cases have been recorded where a gastric ulcer penetrated the substance of the heart and established a communication between the stomach and the left ventricle. The descriptions given by Oser and Chiari, however, seem to refer to the same case.

In three out of the four, definite symptoms of gastric ulcer had existed for a considerable time before the onset of the fatal complication; while in the fourth (Finny) the patient had been liable to severe attacks of pain in the left hypochondrium and below the left clavicle.

The premonitory symptoms of the accident were extremely vague. In one there was a complete absence of pain for a week before death, but the temperature was elevated and heetic in character, and the inspiratory murmur over the root of the left lung was impaired and accompanied by crepitus. In another the patient had suffered from pleurisy, with great pain at the base of the left chest, a few months before.

The initial signs of the accident were the same in all the cases, and consisted of faintness and oppression at the chest. If the leakage was comparatively slight at first, the patient

vomited blood and also passed tarry motions; but when the hæmorrhage was severe and proved rapidly fatal no blood was vomited, and only a small quantity of florid blood was voided by the bowel. This absence of hæmatemesis in violent hæmorrhage into the stomach has already been commented upon (pp. 134, 150, 199).

The duration of life varies with the rapidity of the hæmorrhage; in Finny's case death occurred from collapse within half an hour, but in that recorded by Brenner hæmatemesis and melæna continued for three days.

Case LXIX. A farm labourer, aged 19, was admitted under the care of Dr. Finny into the Sir Patrick Dun's Hospital at the end of October 1885, suffering from subacute articular rheumatism. He was well nourished, though somewhat pale, the knees were painful and swollen, and a pericardial friction sound was heard over the sternum. In a few days the rheumatic symptoms disappeared and the pericardial inflammation abated. The friction sound was not followed by evidences of serous effusion, nor accompanied by endocarditis. The febrile disturbance (99°–101°) which existed at the time of admission diminished, but never disappeared during the five weeks of the patient's residence in the hospital. After the first fortnight he was allowed to sit up, and his diet included eggs, bread, and puddings.

There was no pain in the epigastrium after food, nor any sickness, and there was no history of hæmatemesis. He stated, however, that for the last five years he had suffered pain at intervals over a small area corresponding to the cartilage of the sixth rib on the left side, and also under the left clavicle. At times the suffering was so severe as to oblige him to lie down. Strong pressure with the folded arms always relieved it, and it never became intensified after meals.

While under observation in the hospital he had three or four attacks of this nature, and on two occasions morphine had to be administered for its relief, but during the last week of life it almost entirely disappeared. The fever, however, was much higher, and assumed a hectic type, so that it was thought that probably some latent tuberculosis was present, and this idea seemed to be confirmed by the discovery of imperfect respiration and some crepitus near the root of the left lung. A trace of albumen was found in the urine.

During the night of December 8 the patient had but little sleep; and on the following morning he went to the night chair, complained of feeling weak, and fainted when he was put back to bed. The dejecta consisted of liquid blood. The patient became greatly blanched, and, in spite of immediate assistance, died in half an hour. There was no vomiting.

Autopsy.—The body was intensely anomie. The left pleura contained a little serous fluid, and the layers of the pleura over the diaphragm were adherent. The bronchial glands were enlarged, and one close to the bifurcation of the trachea was caseous and calcified. The pericardial sae was obliterated; the adhesions over the anterior surface of the heart were vascular and recent, and could be easily separated by the finger, but those around the apex and over the posterior surface were old and dense. The valves were healthy.

The stomach and the intestines down to the anus were full of liquid blood, the former alone containing about two quarts. The anterior surface of the stomach was adherent to the diaphragm over an area of 2 inches, but the adhesions were not very dense in character. At this spot, on the interior of the stomach, there was a chronic ulcer $(1\frac{1}{4})$ inches from the lesser curvature. It had perforated all the coats of the viscus, the tendinous portion of the diaphragm, and the obliterated pericardial sac. The floor of the ulcer was rough and granular, and was composed of the muscular structure of the under surface of the left ventricle of the heart, not far from the apex.

No sac or abseess existed between the stomach and the heart; the eommunication was direct, and the size of the exposed portion of the heart was very little smaller than the gastric aspect of the uleer. The eause of the fatal bleeding was not obvious at first, but on passing a probe it was seen that a channel, the size of a No. 5 catheter, existed in the ventricular wall, passing upwards and backwards, and opening into the left ventriele behind, where it was hidden by a musculus papillaris attached to the posterior curtain of the mitral valve.

The muscular structure of the heart was perfectly healthy and free from fatty degeneration, except at the seat of the ulcer, where the fibres were granular, rough, and friable, with several interstices, through one of which the probe passed. (Recorded by Dr. Finny.)

Case LXX. A woman, aged 71, died shortly after her admission into hospital, with the symptoms of gastric ulcer, namely, hæmatemesis and the passage of blood by the bowel. At the autopsy a round hole, 2 centimetres in diameter, was found in the lesser curvature of the stomach. The opening led into a sac as large as a walnut, formed by cicatricial tissue; the sac extended through the diaphragm, pericardium, and the wall of the left ventricle of the heart, and presented at its apex an ulcerated opening large enough to admit an ordinary probe into the left ventricle. The heart was closely adherent to the pericardium over an area 8 centimetres square; the muscular tissue was pale and friable, and had undergone moderate fatty degeneration; the endocardium was thickened for a distance of 1 centimetre round the perforation. In the walls of the sac, close to the opening into the stomach, a hard brittle mass, 2 millimetres long and about 1 milli-

metre thick, was found—probably a piece of glass that had been accidentally swallowed. (Recorded by Chiari.)

Case LXXI. A woman, aged 55, had suffered for several years from attacks of pain in the region of the pracordium, which were sometimes accompanied by vomiting. Six months before her death she had acute pleurisy on the left side, with severe pains radiating into the epigastrium. Three days before death she complained of oppression at the heart, vomited blood, and passed tarry stools. At the autopsy a circular ulcer was found on the lesser curvature of the stomach, which had perforated the diaphragm and communicated with the left ventricle of the heart. (Recorded by Brenner.)

(4) General Emphysema. -- Emphysema of the subcutaneous and subserous tissues of the body is a very rare but interesting result of the perforation of a gastric ulcer. About nine authentic cases have been recorded, the first being described by Cruveilhier. In some instances the subcutaneous emphysema was comparatively slight during life, but became greatly increased after death. In every instance the subserous and subperitoneal tissues were found distended with gas at the autopsy, and as a rule the mediastina were in a similar condition. The gas itself consists principally of hydrogen, as it burns with a blue flame, and is probably a product of gastric fermentation. The manner in which it finds its way into the tissues has been the cause of much speculation. It would appear, however, that the emphysema may arise in two ways. In the case recorded by Faber there can be no doubt that the gastric ulcer perforated into the posterior mediastinum and gave rise to emphysema of the cellular tissue of the thorax, which spread into the neck and afterwards over the whole body. In the other instances, however, the gas would seem first of all to have distended the peritoneal cavity, and subsequently to have found its way into the subserous tissues, either at the edge of the ulcer or at some spot where the peritoneum had been macerated or digested. The accident is invariably fatal, and its general features are well depicted in the following cases.

Case LXXII. A youth about 17 years of age, who had suffered for several months with pain in the belly on exertion, and slight symptoms of indigestion, was suddenly seized with abdominal pain and collapse after indulging somewhat freely in a meal composed of meat and raw turnips. Retching occurred soon afterwards, and he vomited

a large amount of dark brown fluid. When seen about six hours after the onset of the pain, the abdomen was found to be so enormously distended that it appeared as though it would burst. The patient complained of severe spasms of pain and execssive flatulence. The features were pinched, the extremities livid, and the pulse extremely A little later the vomit contained bright blood, and emphysema was detected in the right groin. He then became restless and eyanosed, and lost sensation below the level of the crests of the ilia. He died about twenty-four hours after the appearance of the first symptoms.

Autopsy.—The peritoneal cavity contained a large quantity of gas, and some dark-coloured fluid similar to that which had been vomited auring life. On the posterior wall of the stomach there were two perforations about the size of half a crown. There was extensive emphysema in the cellular tissue surrounding the viscera of the abdomen; and the same was observable in the chest, both lungs in front exhibiting very marked interlobular emphysema. The neck was also swollen with emphysema. The other organs were normal.

(Recorded by Dr. Bell.)

Case LXXIII. A man, 37 years of age, had suffered off and on for several years from indigestion, and for about three years from severe pain after food, vomiting, and constipation. When admitted into the hospital, stenosis of the pylorus resulting from former ulceration was diagnosed, and the stomach was washed out each day. He was discharged somewhat relieved, but died six months afterwards. a few days before death he was troubled with dyspnæa, severe pain, and had ædema of the ankles.

Autopsy.—The skin of the abdomen, chest, neck, face, lower extremities, and left arm was extremely emphysematous. On puncturing it, gas escaped with a whizzing sound, and when ignited burnt with a blue flame. There was much free gas in the peritoneal cavity, and about 4 litres of brownish fluid which looked like the contents of the stomach. The subperitoneal connective tissue was extremely emphysematous, especially in the region of the ascending colon. The diaphragm was pushed up, and bubbles of gas were seen beneath its peritoneal covering. The mediastina were filled with gas, and there was a reddish exudation in both pleural cavities and in the pericardium. The pylorus was thickened and adherent to the gall-bladder, liver, and the hepatic flexure of the colon. The first part of the duodenum was contracted, and immediately below the stricture was a large chronic ulcer. Near the cardiac end of the lesser curvature of the stomach there was another uleer, 2 ems. long and $2\frac{3}{4}$ cms. wide, which had perforated all the coats of the viscus. (Recorded by Poensgen.)

Jürgensen has recorded two cases of sudden death, in

which he found air in the blood-vessels of the body. In one instance severe hemorrhage had taken place, and in the other perforation. His principal case is shortly as follows.

Case LXXIV. A woman began at the age of 39 to experience severe pain at the chest after meals, accompanied by vomiting. After a few months she had an attack of hæmatemesis, which recurred on several occasions during the next six years. When admitted into the hospital she was extremely thin and weak, and markedly anæmic. She complained of intense pain in the epigastrium, which radiated over the greater part of the abdomen and down the thighs. The stomach was slightly dilated, and a sense of resistance could be detected in the epigastrium, which was very tender on pressure. Six days later she had another attack of hæmatemesis, and complained of palpitation and inability to sleep. The hæmorrhage was renewed on the third day, when she became suddenly collapsed. The pulse was irregular, the eyelids slightly ædematous, and the jugular veins greatly distended. Death occurred quite suddenly a few hours afterwards.

At the autopsy the veins of the neck were found filled with air. The abdominal cavity was distended with gas, and there was much emphysema between the stomach and the liver, and in the subpleural and subperitoneal tissues. The cavities of the heart and large vessels, as well as those of the lungs, liver, stomach, and kidneys, were devoid of blood and filled with air. The stomach was constricted in the centre, and upon its posterior wall was a chronic ulcer, 6 or 7 centimetres in diameter. The edges were undermined, and subserous emphysema could be traced from them.

The author regarded the air in the vessels as a product of gastric fermentation which had gained an entrance to them at the edges of the ulcer. It would seem possible, however, that the phenomenon may have taken place to a great extent after death, as neither Oser por Brenner was able to detect air in the heart or arteries after direct perforation of the left ventricle by a gastric ulcer.

(5) Perforation of the Skin (External Fistulæ).—The establishment of an external gastric fistula is a very rare result of the direct perforation of an ulcer. In all the recorded cases the ulcer was very chronic in character, and after contracting adhesions with the abdominal parietes had gradually eroded the tissues. The position of the external opening varies, and may occur either in the left hypochondrium or in the vicinity of the navel, according to the situation of the ulcer. It is always preceded by continued pain over the affected spot for several

months; afterwards a hard tumour forms and the superimposed skin becomes red and shiny. Finally ulceration occurs, and the gastric contents escape from the orifice. When once established, the fistula is permanent. The external opening presents a funnel shape, and the surrounding skin becomes reddened and excoriated by the action of the acid secretion. The principal abnormal symptoms which have been observed. as dependent upon the fistula, are great thirst, increased appetite, obstinate constipation, deficient secretion of urine. and amenorrhea. If vomiting has been urgent before the formation of the fistula, it usually ceases as soon as the chyme commences to escape through the skin. The duration of life varied in the recorded cases from twenty-six days to many years, and in a few death occurred from pulmonary tuberculosis. The diagnosis is easily made by the discovery of food and hydrochloric acid in the discharge from the fistula.

Five cases have been recorded where a chronic ulcer of the stomach perforated the diaphragm, and finally opened upon the surface of the chest through an intercostal space (gastro-thoracic-cutaneous fistula). In most instances the external orifice of the fistula was situated in the eighth, ninth, or tenth left costal interspace, close to the sternum or the ensiform cartilage, but in Middeldorpf's case it was located in the sixth space. All the patients were women, the average age at the time of death being about 41. In every instance the symptoms of gastric ulceration had existed for many years, and the pain had been unusually severe and constant. One patient survived for nearly seven years. The diagnosis of a gastric fistula was easily made by the dribbling away of chyme. In the following case, which has been recorded by Pick, the appearance of a tumour beneath the left breast, combined with the symptoms of gastric ulceration, occasioned a diagnosis of sarcoma of the ribs involving the stomach, but the patient died from acute tuberculosis before the fistula was actually established. It is interesting to observe that no free hydrochloric acid was detected in the gastric contents.

Case LXXV. A woman, 48 years of age, was admitted into the Königsberg hospital for severe pain in the chest and debility. Her history showed that she had suffered from pain after food and vomiting for several years, but that for the last two years these symptoms had been greatly increased, and a tuniour had slowly developed below the

left breast. There was great deformity of the chest from old spinal curvature, but there were no signs of disease in the heart or lungs. The abdomen presented a curious appearance, the right half being sunken and retracted, while the left half of the epigastrium and the left hypochondrium were occupied by a large swelling, over which the peristaltic movements of the stomach were plainly visible. On the left side of the chest, below the breast and between the eighth and twelfth cartilages, was a hard tumour the size of a child's fist, which was adherent posteriorly and to the skin. The costal margin appeared to be much thickened, there was some resistance on palpation over the hypochondrium, and the left lobe of the liver could be felt in the upper part of the epigastrium, and was tender on pressure. The urine contained albumen. For three months the patient remained in the same condition, suffering from intense pain in the abdomen and left side of the chest, with constant vomiting. No free hydrochloric acid was found in the gastric contents, and the superficial tumour did not increase in size. At the end of this time she was attacked with fever, cough, and shortness of breath, and signs of consolidation became evident in the right lung. These symptoms rapidly increased, and she died from exhaustion in about three weeks.

Autopsy.—The fundus of the stomach was adherent to the abdominal wall and to the left wing of the diaphragm. In the pyloric region there was a deep chronic ulcer which extended from the anterior surface across the lesser curvature on to the posterior wall. The anterior part of the ulcer had perforated the coats of the stomach and also the diaphragm to which it was adherent, and communicated by a short track, which led between the cartilages of the eighth and ninth ribs, with the tumour on the chest wall. Examination showed that the latter was composed of inflammatory tissue; and that, had not death taken place, a fistula would soon have been established. There were signs of old phthisis, with recent acute tuberculosis.

(6) Perforation of the Colon.—The formation of a gastro-colic fistula is much more common in cancer of the stomach than in simple ulcer, owing to the rarity of the latter disease in the vicinity of the great curvature. Out of the 33 cases collected by Murchison, 21 were due to cancer and only 10 to ulcer. The simple affection is usually met with between 40 and 55 years of age, and more often in men than in women (5:1).

Occasionally the communication between the two viscera is established without any special subjective symptoms, but in most instances it is marked by sudden pain in the abdomen, diarrhoa and partial collapse, or by facal vomiting. In one of

the recorded cases the patient stated that 'something had burst in his belly.' When the aperture between the stomach and the colon is of moderate size the contents of the bowel find their way into the stomach, and the patient suffers from facal vomiting; but if the hole is comparatively small the ejecta may only possess a feeal odour, or the patient may complain of an intolerable taste in the mouth. Another symptom of great importance is the passage of undigested food by the bowel soon after a meal. It is important to observe that, whenever dilatation of the stomach is present from coexisting stenosis of the pylorus, facal vomiting is usually absent owing to the deficient entrance of food into the intestine; but that lienteric diarrhea, with the evacuation of substances that have just been swallowed, is a striking feature of the case. Reeves was the first to notice that enemata were sometimes vomited if much force was used in their administration. This fact is of considerable value in the diagnosis of a gastro-colic fistula. for if a coloured enema is rejected soon after it has been given, the existence of a communication between the large bowel and the stomach is rendered certain. The passage of the fluid into the stomach is favoured by elevating the lower part of the body during the experiment. Auscultation of the abdomen may reveal a gurgling sound over the site of the fistula (Levinstein).

As a rule the appetite fails, but thirst may be intense. Hæmatemesis and melæna have been recorded in several instances, and if vomiting or diarrhæa is excessive, profound emaciation may occur. In some of the reported cases life was prolonged for several years.

Case LXXVI. A clerk, 54 years of age, who was admitted into the London Hospital after suffering for many years from pain after food and vomiting, was suddenly seized with vomiting of fæces, which were solid in character, and differed in no way from those passed per anum. Subsequently the emesis subsided to a great extent, but whenever the bowels were confined, it immediately became urgent. There was no complaint of pain. These symptoms continued for two and a half years and were accompanied by progressive exhaustion.

Autopsy.—There was firm adhesion between the lower border of the stomach and the transverse colon, the bowel being bent acutely at the point of union. Near the great curvature of the stomach, on its posterior wall, was a chronic ulcer about the size of a shilling, which had perforated the coats of the colon and established a communication between the two viscera nearly three-quarters of an inch in diameter. The nucous membrane of the stomach was thrown into radiating folds round the opening. A cicatrix of a former ulcer was present in the fundus. The pylorus was healthy. (A photograph of the specimen is shown in fig. 29.)

Case LXXVII. A shoemaker, 40 years of age, was admitted into the Brompton Hospital for indigestion and loss of flesh. Fifteen years before he had suffered from severe pain in the stomach and vomiting after meals, and for the last six months he had experienced difficulty of swallowing, and on one occasion had brought up some blood.

A few days after admission the following note was recorded: 'Patient complains of severe headache, with pain in the epigastrium, and sickness. For the last three days, on several occasions he has vomited a quantity of yellow matter having the odour and appearance of relaxed fæces. There is much pain after eating; borborygmi and flatulence; tenderness at the epigastrium, where a sense of firmness can be detected on palpation; extreme anæmia and emaciation. After taking four grains of calomel the patient had several motions, and has vomited as much fæcal matter as he passed per anum.'

A few days afterwards the vomiting ceased, but he complained of an intolerable taste in the mouth, and his breath had the odour of faces. A splashing sound was elicited by percussion over the right side of the abdomen.

Ten days after admission the vomiting recommenced, and the motions were observed to contain much undigested food. These symptoms continued until death took place from exhaustion three weeks after the commencement of the fæcal vomiting. A gastro-colic fistula was diagnosed during life.

Autopsy.—Profound emaciation. Great omentum puckered, and incorporated with the stomach and the arch of the colon, where they were in contact. On tearing it up, a communication between the anterior wall of the stomach (near the pylorus) and the transverse colon was exposed. The orifice in the stomach was about one and a half inches in diameter, and its edges were jagged and thickened. The mucous membrane of the colon was considerably destroyed on either side of the aperture. Fæces were present in the stomach. (Recorded by Dr. Hooper May.)

LITERATURE

DIRECT PERFORATION OF THE PLEURA AND LUNG

Aufrecht, Berl. klin. Woch. 1870, 7, 21. Geiger, cited by Siebert, op. cit.

Günsburg, Archiv f. phys. Heilk., 1852, xi., 3.

Hucbner, Archiv f. Heilkunde, 1871, 3, p. 193.

Kogerer, Prag. med. Woch., 1890, p. 315.

Le Noir, Bull. Soc. Anat., 1890, p. 248.

Müller, Memorabilien, 17, 10, p. 448, 1872.

Needon, Wien. med. Presse, 1869, p. 990.

Ransom, Lancet, 1899, ii. p. 1285.

Rokitanski, loc. cit.

Sieherer, Württemb. Correspondzbl., 1843, xii. p. 26.

Siebert, Casper's Wochenschr., 1842, 29.

Stareke, Deutsche Klinik, 1870, p. 237.

Sturges, Lancet, February 7, 1874.

Tillmanns, Archiv f. klin. Chirurg., 27, 1, p. 103.

PERFORATION OF THE PERICARDIUM

Cérenville, Rev. Méd. de la Suisse Romande, September 1885.

Debove & Renault, Ulcère de l'Estomac.

Fenwick, Lancet, 1897, ii. p. 388.

Guttmann, Berlin klin. Wochenschr., 1880, p. 221.

Moizard, Gaz. Hebdom., 1885, 22, p. 331.

Rosenstein, Ziemssen's Handbuch, Bd. 6, p. 62.

Säxinger, Prager med. Wochenschr., 1865, 1 and 2.

PERFORATION OF THE HEART

Brenner, Wiener med. Wochenschr., 1881, p. 1301. Bruenniche, Hospitalstidende, 1887, 3 R.V.B. p. 697. Chiari, Wien. med. Jahrb., 1880, 30. Finny, Brit. Med. Journ., 1886, i. p. 1102. Oser, Wien. med. Blätter, 1880, 52.

PERFORATION OF MEDIASTINUM

Faber, Württemberg. med. Correspondzbl., 1885, 40.

GASTRO-CUTANEOUS FISTULA

Bineaux, Arch. Gén. de Méd., 1835, ii. sér. tom. 8, p. 214.

Cooke, Western Journ. of Med. & Phys. Soc., Jan. 1834.

Fillenbaum, Wiener med. Wochenschr., 1875, p. 49.

Gooch, Practical Treatise on Wounds, vol. iii. 1774, App. p. 140.

Grunewaldt & Schröder, Ueber den Magensaft des Menschen. Inaug. Dissert.

Dorpat 1853.

Kjönig, Norsk Mag. f. Lægevidensk., 1877, 3. h., vii. p. 589.

Middeldorpf, De Fistulis Ventriculi externis et Chirurgica carum Sanatione Commentatio, 1859, p. 21.

Morgagni, cited by Gérard, Perf. Spont. de l'Estomac, 1803, p. 69.

Murchison, Med. Chir. Trans., vol. 41, p. 11.

Pick, Zeit. f. klin. Med., 1894, 26, p. 452.

Rokitanski, Oesterr. med. Jahrbüch., 1839, 18.

Roux & Corvisart, Journ. de Méd., tom. iii. p. 407.

Streeton, London Med. Gaz., 1829, iii. p. 43.

Wencker, cited by Licutaud, Hist. Anat. Med. liv. 2, obs. 145, p. 327, 1767.

GASTRO-DUODENAL FISTULA

Dittrich, Prager Vierteljahr., 13, p. 125.

Mohr, Casper's Wochenschr., 1842, 16.

Rokitanski, op. cit.

Thierfelder, Deut. Arch. f. klin. Mcd., 1868, p. 33.

GASTRO-COLIC FISTULA

Green, Path. Soc. Trans., 1893, p. 95.

Haller, Opuscula Pathologica, obs. 28, p. 60. 1755.

Levinstein, Schmidt's Jahrb., iii. p. 105.

May, Mcd. Times and Gazette, 1856, p. 38.

Murchison, Edinb. Med. Journ., 1857, vol. iii. p. 121.

GENERAL EMPHYSEMA

Cruveilhier, Anat. Path. t. i. liv. xx.

Demarquay, Essai de Pneumatologic Médicale. Paris, 1866.

Faber, Württemb. med. Correspondenzbl., 1885, 40.

Jürgensen, 'Luft im Blute,' Dcut. Archiv f. klin. Med., 1882, 31, p. 441.

Korach, Deut. med. Woch., 1880, p. 275.

Newman, Lancet, 1868, ii. p. 728.

Bell, Edinb. Med. Journ., vi. p. 783.

Poensgen, Das subcutane Emphysem nach Continuitätstrennungen des Digestionstractus. Inaug. Dissert., Strassb. 1879.

Roger, Archiv. Gén. dc Méd., 1862.

Thierfelder, Deutsches Archiy f. klin. Mcd., 1868, iv. p. 33.

CHAPTER VII

TUBERCULOSIS, CANCER, AND PERNICIOUS ANÆMIA

Phthisis is very common in chronic ulcer of the stomach, nearly 17 per cent. presenting signs of pulmonary tuberculosis after death. The association of the two complaints is usually regarded as merely an accidental occurrence, but a little consideration will show that this supposition is incorrect. In the first place, although both are common diseases they are not equally frequent at the same period of life, tuberculosis being most rife below the age of thirty, while chronic ulcer is chiefly encountered between thirty and fifty. In the second place, phthisis often makes its appearance after the ulcer has healed, especially when the cicatrix has given rise to some deformity of the stomach that impairs the powers of digestion, or has produced a fistula which interferes with absorption. Thirdly, when pulmonary tuberculosis follows gastric ulcer it not infrequently commences at the base of the lung and spreads from below upwards, whereas under ordinary conditions it first involves the apex. Finally, persons who have previously suffered from phthisis are very apt to succumb to a recrudescence of the disease when attacked by gastric ulcer, while acute pulmonary tuberculosis frequently follows extensive destruction of the mucous membrane of the stomach caused by mineral acids or other corrosive poisons.

It is interesting to observe that the gastric and pulmonary complaints are seldom very active at the same time, perforation of the stomach and severe hæmatemesis being very rare when the tubercular mischief is rapidly advancing. It would seem as though a gastric ulcer possessed the same counterirritant properties as are sometimes attributed to fistula in ano.

The various cases which have come under our notice are capable of being divided into three classes. The first includes

those in which evidences of former disease of the lungs, lymphatic glands, or abdominal viscera are found to coexist with recent tubercular mischief. In the second are grouped those less frequent cases where a somewhat rapid form of phthisis supervenes after the gastric complaint has existed for a length of time. In many of them the pulmonary disease commences at the base of the lung. The third class comprises those in which acute phthisis develops shortly after the destruction of the secretory structures of the stomach by corrosive fluids.

- (1) Recrudescent Phthisis.—In more than one half of our cases where recent tubercle was found in the lungs after death evidences of former tubercular mischief were also observed. As a rule, there is an interval of several years between the cessation of the cough and other symptoms of the pulmonary lesion and the development of those which accompany the gastric ulcer; but occasionally the latter ensue without any appreciable interval. Hæmatemesis is rarely observed, but pain after food and vomiting are unusually troublesome. Lossof flesh is also a noticeable feature, and in many instances intercurrent attacks of gastric catarrh are exceptionally frequent. As long as the gastric symptoms continue severe, those which ensue from the recrudescent disease may be so insignificant as to escape attention, and one is often surprised to find recent and extensive consolidation of the lung in a person who complains neither of cough, pain in the chest, nor of night Sooner or later, however, these latter symptoms become troublesome, the pulmonary disease develops rapidly, and death ensues from exhaustion within a few months. Vomiting may continue during the whole time, but the pain after food usually diminishes as soon as the cough becomes pronounced. Hamoptysis and pneumothorax are comparatively rare. Cases of this description are so familiar to every practitioner that there is no necessity to offer any illustrative examples.
- (2) Acute Phthisis.—The sufferers from this variety are principally men of middle age who possess no family tendency to tuberculosis, and who have not suffered from the pulmonary disease previously to the development of the gastric complaint. As a rule the symptoms of gastric ulcer have existed for many years, and have been accompanied by permanent loss of flesh

and strength. Repeated attacks of hæmatemesis also occur in many instances. The pulmonary affection may show itself at any time, but it often does not occur until the cicatrisation of the ulcer has given rise to dilatation of the stomach, to stenosis of the cardiac orifice, or to some deformity of the viscus. When once it has begun it usually pursues a rapid course, and is often attended by severe dyspepsia, vomiting, and constipation. The most interesting cases are those in which it appears to commence as an acute basic pneumonia, which instead of terminating in the usual way pursues a subacute course, and involves the lung from base to apex. This form of the disease has already been incidentally mentioned in several cases, so that a single illustration will suffice.

Case LXXVIII. A gentleman was first seen in 1873, when 47 years of age. His father had suffered from hæmatemesis, but had lived to old age; one of his sisters had died from some disease attended with uncontrollable vomiting. He had first been attacked with violent pain in the abdomen supposed to be eolie, and ehiefly complained of severe pain in the epigastrium coming on shortly after food. There had been no vomiting or hæmatemesis, but he had lost a eonsiderable amount of flesh. He gradually improved, so that in 1874 he was able to take ordinary diet without pain. Up to the year 1893 he was frequently under observation for pain after food, distension, flatulence, and constipation of the bowels. He then suddenly passed blood by stool, which was followed by severe hæmatemesis. After his recovery he remained liable to pain after food and other symptoms of dyspepsia, and continued thin and weak. In January 1899, being 73 years of age, he was attacked with cough, expectoration, and fever. He still complained of pain shortly after food. There was well-marked dulness at the base of the right lung, with tubular breathing and crepitation. The consolidation spread from below upwards, tubercle bacilli were found in the sputa, and he gradually sank, the temperature remaining high until his death. On post-mortem examination three well-marked sears were discovered in the region of the pylorus, and an ulcer the size of a shilling was situated near the cardiac orifice. The whole of the right lung was affected with tubercular consolidation, with small cavities here and there. The opposite lung was also diseased.

(3) Acute Phthisis following destruction of the Stomach.— It is probable that whenever the powers of digestion and assimilation are suddenly and seriously injured, the state of malnutrition that is engendered so reduces the natural powers of resistance to infection as to permit the invasion of the tubercle bacillus. The condition is a rare one, so that the following cases are endowed with exceptional interest.

Case LXXIX. A potman, 34 years of age, was admitted into the London Hospital on October 16, 1888, with the symptoms of acute irritant poisoning, having swallowed some oxalic acid about an hour

previously.

After the administration of the usual remedies the stage of collapse gradually passed off, but the patient complained of a burning pain at the epigastrium attended with frequent retching. During the next twenty-four hours the pain and vomiting continued severe, and the ejecta on several occasions contained traces of altered blood. On the third day of his illness he had an attack of melæna, and this symptom recurred several times.

For a fortnight after taking the poison the patient continued to exhibit the symptoms of severe inflammation of the stomach, violent attacks of vomiting ensuing on every attempt to swallow any form of food.

On November 5, nineteen days after the commencement of the disease, it was noted that the patient had only been sick twice during the preceding forty-eight hours, and could now swallow small quantities of liquid nourishment. There was still considerable pain over the region of the stomach, increased by pressure with the hand.

On November 10 vomiting again became urgent, the ejecta consisting of an exceedingly sour-smelling fluid which contained a large quantity of lactic acid but was devoid of any trace of free hydrochloric acid. Numerous torulæ and bacteria were detected with the

microscope.

One month after admission it was noted that the patient vomited once or twice a day, about 20 ounces of a sour fluid, devoid of free hydrochloric acid, being ejected on each occasion. There was still considerable pain experienced at the epigastrium after taking liquid food. The stomach was found to extend one inch below the navel, and pressure over the pyloric region gave rise to pain.

A fortnight later, on November 27, the dilatation of the stomach was more apparent, and the amount of the daily vomit measured

70 ounces. No free hydrochloric acid could be detected in it.

By December 10 the patient was sufficiently recovered to leave his bed for a few hours daily. He was now able to take small quantities of bread and milk and similar food without vomiting, but the more solid forms of nourishment gave rise to immediate pain at the epigastrium, and were speedily rejected. The body weight, which on admission was said to have exceeded 10½ st., was now only 7 st. 8 lbs.—a loss of rather less than 3 stones. The temperature remained steadily at a point somewhat below the normal, the tongue was flabby and indented by the teeth, and the bowels were obstinately eonfined. The stomach was now found to extend $2\frac{1}{2}$ inches below the umbilieus, and to present a well-marked splash, which was audible to the patient himself when he turned over in bed. Under these circumstances it was determined to wash out the organ with warm water, the operation being performed by means of a glass funnel and a soft tube.

The daily employment of lavage, combined with eareful feeding, brought about a rapid improvement in the general condition. A month later, on January 14, the body weight had increased by 7 lbs. The patient still complained of attacks of severe pain at the epigastrium, occurring at irregular intervals but always aggravated by the ingestion of food. The vomiting had also diminished in severity, and several days sometimes elapsed between the attacks. On pumping air into the stomach by means of a hand bellows the lower border of the organ was found to extend about 2 inches below the umbilicus. No trace of free hydrochloric acid could be discovered in the fluid extracted from the stomach.

On January 25 the patient complained of feeling chilly, and thought he had caught a cold while walking in the garden. The following week it was noted that he was not so well. He had a troublesome cough attended with slight expectoration, and was much disturbed at night by profuse perspirations. The temperature also, which previously had remained constantly below the normal point, now registered 101° F. in the morning, and 102.5° F. at night. The vomiting was very troublesome, and the steady increase in weight was not maintained. The physical signs denoted the existence of some general bronehitis.

A fortnight later, on February 13, the body weight had decreased by nearly 7 lbs. The patient was very weak, and troubled by constant cough and expectoration. Comparative dulness on percussion was detected at the right apex, with numerous moist crepitations on inspiration.

From this time onward the pulmonary condition rapidly developed. At the commencement of March the upper part of the right lung was comparatively dull as far as the fourth rib, with the auscultatory signs of consolidation; while an impaired note at the left apex and right base posteriorly, with abundant moist sounds, gave evidence of the wide-spread character of the disease.

On March 8, the patient had a sharp attack of hemoptysis. The expectoration was found to contain numerous tubercle bacilli. The temperature seldom declined below 103°, and occasionally registered over 104°. Along with the development of the pulmonary disease

the gastric condition became worse. Vomiting now occurred frequently, and the spasmodic attacks of pain required the constant exhibition of morphine for their relief. The patient's strength rapidly failed, and he eventually succumbed to extreme exhaustion on April 5, 1889, rather less than six months after taking the oxalic acid, and about six weeks after the onset of the pulmonary symptoms.

At the post-mortem examination the lungs were found to be the scat of an acute tuberculous process which involved their whole tissue from apex to base. In the upper lobe of the right lung there was a cavity the size of a large walnut. The stomach was greatly dilated, and contained 54 oz. of fluid. In the cardiac and middle zones the mucous membrane exhibited a peculiar glistening appearance, and was firmly adherent to the muscular coat, the whole wall of the organ being remarkably thin and transparent. Radiating over the surface were a number of fibrous bands of the nature of superficial cicatrices. The mucous membrane in the pyloric region was abnormally thick, and presented the characteristic features of the ctat mamelonné. The pyloric orifice had been narrowed by the thickening and contraction of the mucous membrane in its immediate neighbourhood, and would only admit the passage of a small catheter. On microscopic examination the mucous membrane in the cardiac and middle thirds of the organ was found to have been converted into a layer of fibrous tissue a few lines in thickness, in which little or no indication of the former structure of the stomach could be detected. The other organs were normal.

Case LXXX. A man, æt. 32, came under treatment in May, 1886, for a chronic disorder of the stomach.

He stated that when abroad some five months previously he had accidentally swallowed some nitric acid, and had been immediately seized with great pain in the abdomen and vomiting. As the result of this accident he had been forced to keep his bed for nearly two months, and for many weeks had been unable to take any form of nourishment by the mouth without vomiting.

He now complained that whenever he attempted to swallow solid food it appeared to stick in the gullet and gave rise to pain at the lower part of the chest on the left side, and was usually vomited within a short time. When he swallowed a mouthful of liquid the fluid appeared to trickle slowly into the stomach past some obstruction, but if he attempted to drink quickly the major part of it regurgitated at once. Even when food did reach the stomach it invariably caused considerable pain at the epigastrium, and was often rejected. On several occasions, especially at the beginning of his illness, he had vomited blood, the last attack having occurred five weeks previously. The bowels were obstinately confined. Before the commencement of the disorder he had always enjoyed excellent health, and

as far as he was aware none of his family had suffered from phthisis.

On examination the patient was found to be extremely anemic and emaciated, and according to his own statement he had lost nearly 4 st. in weight. The pulse was small and feeble, the temperature 97°, and the tongue covered with a thick fur. The breath was very sour and offensive. The stomach was considerably dilated, and the lower border extended two fingers' breadth below the level of the umbilicus. The peristaltic movements were faintly visible, and a well-marked splash was obtained on palpation. The whole of the epigastric region was somewhat tender, and pressure over the pyloric extremity caused pain. The other organs were normal.

The distinct history of corrosive poisoning, coupled with the symptoms and physical signs just narrated, seemed to point to a constriction at the lower end of the œsophagus along with chronic gastric catarrh and stenosis of the pylorus. It was accordingly determined to wash out the stomach regularly and to administer food by means of the tube. On attempting to pass a soft tube some resistance was encountered at a spot about 42 cm. from the incisor teeth, but this was easily overcome and the stomach thoroughly washed out. The fluid extracted from the organ was yellowish-brown in colour, and very sour-smelling. It contained a large quantity of lactic acid, but appeared devoid of free hydrochloric acid. Lavage was afterwards performed daily, and the patient soon learned to accomplish it for himself. The effect of keeping the stomach clean and administering food through the tube soon became apparent; the pain and vomiting were considerably relieved and the patient rapidly gained in weight.

Three months later the patient had increased his weight by more than a stone. He still complained of attacks of pain in the region of the stomach, which were aggravated by the ingestion of food, but the vomiting was much less troublesome. On examination the epigastrium was still tender, especially over the pyloric region of the stomach. The stomach itself showed signs of considerable dilatation, and on being distended with air the lower border reached several inches below the navel. No free hydrochloric acid could be detected in its contents.

Six weeks later (nine and a half months after swallowing the acid) the patient expressed himself as feeling much worse. For a fortnight he had been troubled with a severe cough and expectoration, and had lost several pounds in weight. At night his sleep was disturbed by profuse sweats, and each paroxysm of the cough brought on an attack of vomiting with severe pain at the epigastrium.

The temperature now registered 102°, the pulse was quick, and the patient seemed to suffer from some dyspnæa. The stomach was

found to extend as low down as a spot midway between the umbilicus and the pubes, and presented a loud splash. The percussion note over the upper part of the left chest was comparatively dull as far as the fourth rib, and coarse moist sounds were audible over this area. The percussion note was also defective at the right base posteriorly, and numerous rhonchi with occasional crepitations were to be heard scattered over the whole chest.

From this time the pulmonary phenomena pursued an acute course. Tubercle bacilli were demonstrated in the expectoration, the gastric symptoms became greatly aggravated, and the patient succumbed seven weeks after the first appearance of the phthisical symptoms, and rather more than ten months after taking the nitric acid.

At the autopsy both lungs were found to be studded with caseous tubercle, which in many places had already broken down into small cavities, notably in the left upper lobe. The stomach was enormously dilated, and occupied the greater part of the anterior aspect of the abdominal cavity. At the lower end of the œsophagus, for about 7 inches, the mucous membrane showed evidences of longitudinal scarring. About $1\frac{1}{2}$ inches from the cardiac orifice the tube was distinctly dilated and its wall thickened, while just above its termination there existed a cicatrix, involving a quarter of its circumference and causing considerable narrowing of its lumen. The stomach contained a large quantity of fluid and gas. In the fundus of the organ the mucous membrane had undergone post-mortem digestion, but elsewhere the whole of the secreting surface was found to have been completely destroyed, and a thin layer of fibrous tissue was all that remained to represent the mucous membrane. Close to the pylorus and situated on the lesser curvature there was a chronic ulcer about the size of a florin. The edges were thick and irregular, and the base was formed of the muscular coat of the organ. The contraction of the edge of this ulcer had produced a puckering of the surrounding tissues, and so contracted the pyloric orifice that it would only admit the introduction of a lead pencil. The other organs were normal.

Robert 1 gives the details of a case the main features of which are almost exactly similar to the preceding. The patient, a man 32 years of age, was attacked with the symptoms of severe inflammation of the stomach after swallowing some nitric acid. The organ was found to be much dilated, and he suffered from several attacks of hæmatemesis. At the end of ten months he suddenly developed a cough accompanied by fever, and soon exhibited all the signs of acute phthisis. At the end of six weeks he succumbed to pneumothorax.

¹ Bull. Soc. Anat. 1880, p. 309.

At the autopsy a chronic ulcer was found in the pyloric region of the stomach, causing contraction of the orifice. Elsewhere the inner surface of the organ presented a smooth shining appearance, with cicatricial bands radiating in all directions. Both lungs were profusely studded with the so-called broncho-pneumonic tubercle, and a small cavity in the left upper lobe had ruptured into the pleural sac.

The three cases just cited are almost identical in their clinical and pathological features. In each case the corrosive fluid had given rise to severe inflammation with subsequent destruction of the secreting surface of the stomach. The onset of the pulmonary disease was always heralded by a sudden elevation of the temperature, which previously had remained constantly below the normal point. Although none of the patients possessed a family tendency to phthisis, the disease in each case ran a very acute course, extending over a period of about six weeks. It is also worthy of remark that in the two instances where the vomit was systematically examined no trace of free hydrochloric acid could ever be detected.

Cancer.—It is a well-known fact that carcinoma is apt to attack the edges or scar of a simple ulcer, but the frequency with which this complication occurs is probably overestimated. Only two genuine examples were observed in the post-mortem room at the London Hospital during a period of forty years, and only three cases of the kind are recorded in the Transactions of the Pathological Society of London. Kollmar in 1891 was able to collect about fourteen authentic examples. On the other hand, several Continental writers speak of carcinoma as a frequent complication of gastric ulcer, and Bouveret states that he had seen three cases of the kind. There is always a great difficulty, as Dittrich originally pointed out, in distinguishing a thick cicatrix from a scirrhous deposit in the stomach, and it has already been mentioned that chronic ulcers with irregular and everted edges are very liable to be mistaken for new growths, so it is possible that many cases have been recorded as cancer from their naked-eye appear-This supposition is corroborated by two instances related by Kollmar, where it was only after microscopic examination that the benign nature of the disease was determined.

Cancer may develop in an ulcer as a primary growth, or occur as a secondary deposit in cases of carcinoma of the breast

TUBERCULOSIS, CANCER, PERNICIOUS ANÆMIA 379

or other organ. In one of our cases two masses of scirrhus were found in the edges of a chronic ulcer in a man who had died from cancer of the skin of the chest. Secondary deposits are found in the liver in rather more than half the cases, and in some of the recorded instances it was the discovery of these secondary nodules that led to a microscopic examination of what had been pronounced to be a simple chronic ulcer, and the consequent detection of a cancerous growth in its base (Hauser).

The disease is rather more common in women than in men, and also commences in them at a somewhat earlier age. The symptoms vary according as the disease attacks the edge of an ulcer or invades its scar.

When the ulcer itself is attacked by cancer it may usually be observed that the appetite diminishes, the pain after food increases and becomes more continuous, while emaciation makes rapid progress. As a rule the growth occurs near the pylorus, so that the symptoms of obstruction at the outlet of the stomach often develop with some rapidity. In all cases a true cachexia gradually appears, hæmorrhages occur at intervals, and the complaint runs a rapid course. In those cases where a cicatrix is invaded by carcinoma the patient gives a history of former ulceration, the symptoms of which had gradually subsided before the onset of the secondary complaint. The pain that accompanies the malignant disease is more continuous and more severe than that of the benign affection, is not materially influenced by diet, and is accompanied from the first by rapid loss of flesh and strength, while the appetite disappears and cachexia develops.

In about two-thirds of the cases a tumour may be detected in connection with the stomach after the lapse of a few months. It is distinguished from that which occasionally ensues from simple ulcer by its rapid increase in size, its irregular outline, and its tenderness on pressure (p. 208).

The condition of the gastric secretion varies according to the state of the former ulcer. When this remains unhealed hypersecretion usually continues throughout the whole course of the disease [Bouveret, Rosenheim], or the free hydrochloric acid, which was previously in excess, gradually diminishes and finally disappears [Dieulafoy]. If, however, the cancer has attacked a scar which had not involved the pylorus, the free

acid usually disappears at an early stage of the malignant disease.

The diagnosis is always difficult, more especially in cases where the ulcer has long been accompanied by debility and cachexia, or by chronic gastritis with an absence of free hydrochloric acid [Kollmar]. The presence of a growing tumour at the pylorus, and of secondary deposits in the liver or peritoneum, is the only certain sign of its existence.

The disease usually terminates fatally within six months.

Pernicious Anæmia.—It is often stated that chronic ulcer of the stomach is very apt to be followed by idiopathic or pernicious anæmia. The fact that Rosenheim and other observers of repute have recorded cases in which this sequence of events occurred, is sufficient testimony to their occasional co-existence; but that true pernicious anæmia is a frequent sequela of the gastric complaint is more than doubtful, and we have never met with a genuine example of it either in private or hospital practice. On the other hand, from the numerous cases that have come under our notice which were supposed to be suffering from the two affections, we are inclined to believe that in the majority the symptoms of anæmia are due to one or other of the following conditions: (1) Undetected hæmorrhage; (2) Excessive hæmorrhage; (3) Cicatricial deformities of the stomach or duodenum; (4) Unsuspected cancer.

(1) Undetected Hamorrhage.—In about two-thirds of the entire number it was definitely ascertained that the progressive anæmia was due to hæmorrhage from the bowel which had escaped attention. In some instances careful inquiries elicited a history of sudden attacks of faintness and diarrhea, accompanied by black stools; but as a rule the existence of the melæna had to be determined by actual investigation. The condition is chiefly encountered in men from 40 to 50 years of age, who have suffered from vague symptoms of indigestion but have never vomited blood, and as a rule the autopsy shows that the ulcer is situated in the duodenum. The patients usually seek advice on account of increasing debility and breathlessness, and, in the absence of definite symptoms pointing to ulcer of the stomach, the practitioner is apt to concentrate his attention upon the obvious anæmia. Another point of resemblance between this symptomatic affection and the pernicious variety is that from time to time attacks of sickness

and diarrhea occur, accompanied by a rise of temperature and a notable increase of the general symptoms. A correct diagnosis can be made by constantly examining the evacuations, especially during the periods of exacerbation, when the so-called diarrhea is found to consist almost entirely of black blood. It is also to be observed that, although the red corpuscles in the circulation are greatly diminished in number, they never exhibit any characteristic changes in shape and size. The following case was supposed to be a good example of pernicious anemia until the discovery of the intestinal hemorrhage.

Case LXXXI. A gentleman, 42 years of age, who had suffered for about fifteen months from dyspepsia and debility, was attacked while taking a walk on a hot day with faintness, sickness, and diarrhæa. He soon recovered, but felt weak and unenergetic, and suffered much from excessive flatulence. About three months later he suddenly fainted after a ride on horseback, and had several loose actions of the bowels. He then complained of great weakness and breathlessness, and palpitation ensued upon the slightest exertion. The doctor who was called in found him very anæmic and feeble, but could detect no signs of disease in the various organs of the body. Under the use of iron the patient improved somewhat, but in about six weeks he had another attack of sickness and diarrhæa which lasted four days and left him very prostrate. The temperature of the body was elevated at night, the appetite disappeared, and the pallor was so intense that the case was diagnosed as pernicious anæmia.

When we saw him a fortnight later he was confined to bed and so fceble that he could hardly raise his head from the pillow. The pulse was weak, 100 per minute, the respiration 24 per minute, and the tongue coated with a brown fur. The skin was dry, but there were no signs of any rapid loss of flesh. The temperature at nights was 100.5, and in the mornings about 99° F. There was no cough or complaint of pain, the bowels were very confined, and he suffered from frequent belching of wind. The skin and mucous membranes were intensely anæmic, and the face exhibited a pale lemon tint. There was no jaundice. The urine was scanty and free from albumen, sugar, and bile. A loud hæmic bruit was audible over the base of the heart, but there were no signs of disease of the thoracic organs. The abdomen was slightly distended, and the lower border of the stomach extended rather more than an inch below the level of the navel. There was some tenderness on pressure two inches above and to the right of the umbilieus, but no tumour could be detected. The red corpuscles of the blood were greatly diminished (56 per cent.). On careful questioning the patient admitted that he had long been troubled with indigestion, and oecasionally had experienced violent pain in the right side of the abdomen about five in the afternoon, which was sometimes followed by vomiting. There had never been any blood in the vomit, but as he never looked at his motions he could not describe their character during the attacks of diarrhæa. He had always been a temperate man, had not had syphilis, and had never been troubled with piles. When the bowels acted in response to an enema the stool was found to contain a large quantity of altered blood. We accordingly suggested that the anæmia was due to repeated hæmorrhages from a duodenal ulcer, and recommended a milk diet with suitable styptics and absolute rest in bed. Under this treatment the patient gradually recovered.

(2) Excessive loss of Blood.—When a patient with a gastric ulcer has frequent attacks of severe hæmorrhage, the loss of blood is often inadequately repaired, and he consequently suffers from profound anæmia, attended by extreme debility, dyspnæa on exertion, discomfort and flatulence after meals, and perhaps cedema of the ankles. The percentage of red corpuscles in the blood is greatly reduced, and both microcytes and macrocytes may occasionally be observed. The gastric contents may also lack free hydrochloric acid. This condition often terminates fatally and was responsible for the diagnosis of pernicious anemia in many of the recorded cases. As far as we know, however, the existence of atrophy of the gastric mucous membrane has never been demonstrated after death; nor yet the presence of iron-containing pigment in the liver and spleen. The following case recorded by Zahn probably belonged to this category, although it is always cited as an example of pernicious anæmia. Its chief interest lies in the coexistence of esophageal and duodenal ulceration.

Case LXXXII. A man, 46 years of age, who had led a very intemperate life, began to lose his appetite, and to suffer from pain in the epigastrium and vomiting after food. After the lapse of some months he had an attack of bæmatemesis, which recurred several times. When admitted into the hospital he was very thin and intensely anæmic, the skin being of a pale lemon colour. The pulse was small and feeble, and a loud hæmic bruit was audible over the base of the heart. Pressure over the epigastrium gave rise to pain, but no tumour could be detected. Under treatment he gradually improved, but after returning home he was again seized with pain and hæmatemesis, and was re-admitted into hospital in a dying state. The anæmia was

then profound and the number of red corpuscles in the blood only 950,000 per cubic mm. At the autopsy, a chronic ulcer was found at the lower end of the œsophagus and another in the duodenum. There was some fatty degeneration of the heart.

- (3) Deformities of the Stomach from Cicatrisation.—It has already been stated that the various deformities of the stomach which result from simple ulcer are sometimes accompanied by anæmia and debility, while under certain conditions the principal symptoms of the ulcer itself are extreme cachexia and loss of flesh. In both conditions the mucous membranes lose their colour, and the patient suffers from dyspnæa, palpitation, and ædema of the feet after exertion. The appetite disappears, vomiting occurs at intervals, and the corpuscular richness of the blood gradually diminishes. As a rule, however, the temperature of the body is subnormal, the characteristic changes in the red corpuscles are absent, and the gastric contents exhibit an excess of free hydrochloric acid.
- (4) Cancer of the Stomach.—In a large proportion of the cases that are diagnosed as ulcer with pernicious anæmia, the patient is really suffering from cancer of the stomach. In some of our cases of this description the patient was unusually young (25 to 40), and the symptoms of severe pain after food and vomiting had been quickly followed by those of intense anæmia. There was seldom any palpable tumour in connection with the stomach, and for some time we were often in doubt as to the exact nature of the disease. After a few months, however, the continued loss of flesh and strength, the presence of altered blood in the vomit, or the disappearance of free hydrochloric acid from the gastric contents and of sulphocyanide of potassium from the saliva, indicated the existence of gastric carcinoma. When cancer following simple ulcer is accompanied by profound anæmia its recognition is more difficult.

With regard to the supposititious influence of gastric ulcer upon the development of peripheral neuritis, endocarditis, abdominal tuberculosis, renal disease, and other maladies, we have no evidence to offer.

LITERATURE ON CANCER SECONDARY TO ULCER

Biach, Wien. med. Presse, 1890, 13, p. 488.

Bouveret. Maladies de l'Estomac, Paris 1893, p. 274.

Brinton, Diseases of the Stomach; Path. Soc. Trans., vol. ix. p. 209.

Cruveilhier, op. cit.

Debove and Renault, Ulcère de l'Estomac.

Dienlafoy, La Presse Médicale, November 10th, 1897.

Dittrich, Prager Vierteljahrsch., 1848, vol. xvii. p. 1.

Dreschfeld, Medical Chronicle, vols. xiii.-xv.

Eisenlohr, Deut. med. Wochen., 1890, p. 1243.

Flatow, Ueber die Entwicklung des Magenkrebses aus Narben des runden Magengeschwüres. Inaug. Dissert., München 1887.

Goodhart, Path. Soc. Trans., vol. xl. p. 78.

Hauser, Das chronische Magengeschwür, 1883.

Hayem, La Presse Médicale, August 4, 1897.

Heitler, Wiener med. Wochen., 1887, 31.

Hemmeter and Ames, Medical Record, September 11, 1897.

Hiekman, Path. Soc. Trans., vol. l. p. 107.

Kollmar, Berl. klin. Wochen., 1891, 28, p. 119.

Kuleke, Zur Diagnose und Therapie des Magenearcinoms. Dissert., Berlin, 1889.

Lebert, Die Krankheiten des Magens, p. 341.

Mayer, Ein Fall von Uleus simplex in Verbindung mit Carcinom. Dissert., Berlin, 1874.

Pitt, Path. Soc. Trans., vol. xlvi. p. 82.

Rokitanski, op. cit.

Rosenheim, Berl. klin. Wochen., 1889, p. 1031.

Werner, Württ. med. Corresp., 1869, p. 1022.

Acute ulcer of stomach, age in, 81, 131 ABDOMINAL WALL, perforation of, 64, appearances of, 13 diagnosis of, 159 Abdominal tumour from abscess, 336 diet in, 167 adhesions, 209 drugs in, 169 ulcer, 208 Abscess, periduodenal, 65, 323 etiology of, 78 frequency of, 78 boundaries of, 65 from burns, 87 diagnosis of, 345 frequency of, 65 signs of, 334 in cardiac diseasc, 151 in fevers, 146 in liver disease, 154 symptoms of, 334 terminations of, 66, 334 in pyæmia, 146 treatment of, 349 hæmatemesis in, 132 local peritonitis from, 143 varieties of, 337 Abscess, perigastric, 58, 323 melæna in, 134 microscopical features of, 25 acute, 324 pain in, 138 boundaries of, 59 chronic, 327 perforation of, 139 primary, 131 complications of, 62, 341 diagnosis of, 345 prognosis of, 165 frequency of, 58 recurrence of, 165 latency of, 327 scars from, 10, 35 secondary, 146 sequelæ of, 165 signs of, 334 symptoms of, 324, 327 terminations of, 63, 341 sex in, 80 treatment of, 349 sudden death from, 141 surgical treatment of, 175 varieties of, 324 Abscess, subphrenic, 58, 323 symptoms of, 131 Acetonuria in ulcer, 206 treatment of, 167 Acidity, causes of, 202 varieties of, 131 treatment of, 251 voniting in, 138 Acute paralysis of stomach, 319 Adhesions, periduodenal, 30, 269 Acute plithisis following ulcer, 371 cause of, 30 Acute ulcer of duodenum, 156 frequency of, 31 appearances of, 16 results of, 31, 331 Adhesions, perigastric, 28, 269 etiology of, 78 frequency of, 79 causes of, 28 from burns, 87, 158 dangers of, 31, 269 hæmorrhage in, 157. diagnosis of, 272 perforation of, 157 frequency of, 28 results of, 31, 269 signs of, 157 signs of, 270 situation of, 12 symptoms of, 269 size of, 4 treatment of, 275 symptoms of, 156 treatment of, 167, 175 tumour from, 209 Acute nleer of stomach, 16, 131 varieties of, 269 acute peritonitis from, 142 Age in duodenal ulcer, 84

Age in gastric ulcer, 81 Air in blood-vessels, 363 Alimentation, reetal, 171 subcutaneous, 172 Amenorrhea from hemorrhage, 205 in acute ulcer, 94 chronic ulcer, 205 Anæmia as cause of non-healing, 128 from hæmorrhage, 166, 380 in acute uleer, 93 in chronie ulcer, 204 in deformities of stomach, 222 in hypersecretion, 303 pernicious, 380 Anæmic fever, 137 Aneurism of duodenal arteries, 53 of gastric arteries, 24 fatal hemorrhage from, 24 Annular ulcer, 13 Antiseptics, uses of, 251, 282, 309 Aorta, perforation of, 53 Ardent spirits as cause of ulcer, 86 Arsenic, employment of, 254 Astringeuts, uses of, 249

BACTERIAL NECROSIS, theory of, 114 Belt, abdominal, uses of, 275, 280 Bibliography of duodeual ulcer, 262 cancer, 383 gastric fistulæ, 367 gastric ulcer, 255 hour-glass stomach, 298 hypersecretion, 310 perigastric abscess, 349 surgical treatment, 266 tetany, 321 Bile duct, obliteration of, 295 jaundice from, 296 symptoms of, 296 treatment of, 297 Bismuth salts, administration of, 249 Blood, appearances of, in hamatemesis, 135, 195 Blood, state of, in chronic ulcer, 205 Blood-vessels, air in, 363 aneurisms of, 24 eroded by gastric ulcer, 50 by duodenal ulcer, 52 Blood, vomiting of, 159 Burns as cause of duodenal ulcer, 87 gastric ulcer, 109

Cachecric variety of ulcer, 221
diagnosis of, 238
Cachexia with cancer, 379
Cancer following ulcer, 75
frequency of, 75, 378
symptoms of, 379
varieties of, 378
Cardia, acute ulcer of, 151

Cardia, chronic ulcer of, 11 Cardia, stenosis of, 11, 283 symptoms of, 287 treatment of, 290 varieties of, 288 Cardiac disease with ulcer, 76, 151 Carlsbad salts, uses of, 252 Catarrh of stomach from ulecr, 74 symptoms of, 202, 303 Catarrhal variety of ulcer, 218 Children, gastric ulcer in, 83 Chronic ulcer of duodenum, 4, 183 bibliography of, 262 diagnosis of, 241 etiology of, 78 morbid anatomy of, 1 sequelæ of, 269 symptoms of, 212 treatment of, 243 Chronic ulcer of stomach, 1, 183 acetonuria in, 206 acidity in, 202 age in, 81 amenorrhæa in, 205 anæmia in, 204 antiseptics iu, 251 aperients in, 252 appetite in, 203 arsenic in, 254 astringents in, 249 bibliography of, 255 bismuth in, 249 blood condition in, 205 cachectic form of, 221 cachexia in, 205 Carlsbad salts in, 252 cause of death in, 226 clinical varieties of, 215 constipation in, 203 diagnosis of, 230 diarrhæa in, 203 diet in, 243 duration of, 224 dyspepsia in, 202 dyspeptic variety of, 202 etiology of, 78 flatuleuce in, 202 frequency of, 78 gastralgic form of, 215 gastric secretion in, 212 hæmatemesis from, 195 hæmorrhage from, 193 hemorrhagic form of, 221 hyperacidity in, 212 hypersecretion after, 299 iron in, 253 latency of, 220 loss of flesh in, 204 morbid anatomy of, 1 mortality in, 226 onset of, 183 pain in, 184

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Chronic ulcer of stomach, pathogenesis	
of, 97	nervous dyspepsia, 236
perforation of, 200	nervous vomiting, 235
posture in, 188	pernicious anæmia, 239
prognosis of, 225	Diagnosis of duodenal ulcer, 241
rectal feeding in, 243	gastro-colic fistula, 366
saliva in, 204	hæmatemesis, 160
sedatives in, 248	hour-glass stomach, 295
sequelæ of, 269	perforation of diaphragm, 356
signs of, 206	periduodenal abscess, 341
silver salts in, 249	perigastric abscess, 334
symptoms of, 184	perigastric adhesions, 272
temperature in, 205	pneumo-pericardium, 357
thirst in, 203	pyo-pneumothorax, 347
tongue in, 203	stenosis of cardia, 288
tumour from, 208	stenosis of bile duct, 295
treatment of, 243	stenosis of pylorus, 280
urine in, 206	Diaphragm, perforation of, by abscess,
vomiting in, 191	64, 343
Cicatrisation of duodenal ulcer, 35	by ulcer, 68, 351
results of, 295	Diaphragmatic hernia, 70
gastric ulcer, 33	Diet in acute ulcer, 167
frequency of, 33	chronic ulcer, 244
process of, 37	dilatation of stomach, 282
results of, 38, 277	gastric catarrh, 310
Cicatrix, nature of, 35	hypersecretion, 308
tumour from, 211	Digestion in hypersceretion, 306
cancer of, 75 , 379	Dilated stomach from adhesions, 271
Cirrhosis of liver in ulcer, 76, 154	from pyloric stenosis, 277
Cirrhosis of stomach from ulcer, 74	from ulcer, 38
Clinical varieties of acute ulcer, 131	Diseases of stomach in ulcer, 74
of chronic ulcer, 215	Diseases of viscera in ulcer, 75
Climate, effects of, 78	Diverticula of duodenum, 47
Colon, perforation of, by abscess, 64, 344	of stomach, 46
by ulcer, 71, 365	Dorsal pain, 186
Conclusions about pathogenesis, 128	Duodenum, abscess connected with, 65,
Congestive theory, 104	323
Constitutional causes of non-healing, 128	acute ulcer of, 156
Constipation, causes of, 202	chronic ulcer of, 212
treatment of, 252	diverticula of, 47
Corrosive poisoning, ulcer from, 113	hæmorrhage from, 52, 213
phthisis after, 372	perforation of, 58, 214
Crenate ulcer, 14	scars in, 35
Crescentic ulcer, 11	stenosis of, 45
	Duration of ulcer, 224
	Dyspepsia from ulcer, 166, 202
Dangers of rectal feeding, 170	Dyspeptic variety of ulcer, 220
Deformities of duodenum, 45	
of stomach, 38, 277	ELECTRICITY in diagnosis, 188
Diacetonuria, 206	Emboli in stomach, 101
Diagnosis of acute gastric ulcer, 159	Embolism as cause of ulcer, 100, 102
Diagnosis of cancer with ulcer, 380	Empyema from ulcer, 352
Diagnosis of chronic gastric ulcer, 230	Emphysema after perforation, 70, 361
from cancer, 238	Endarteritis, 103
chronic gastritis, 235	Endocarditis, 76
duodenal ulcer, 241	Enemata, nutrient, 170, 172
gall stones, 232	Enteric fever, ulccration in, 148
gastralgia, 231	Epigastric pain, 184
gastric hyperæsthesia, 231	pulsation, 207
hyperacidity, 230	reflexes, 207
hypersecretion, 230	temperature, 207
locomotor ataxia, 233	tenderness, 187

114	191914
Epilepsy, 311, 316	Gastritis from hypersecretion, 299
Erosion of blood-vessels, 49	symptoms of, 303
Erysipelas, ulcers in, 148	treatment of, 309
Etiology of gastric ulcer, 78	Gastritis, chronic, diagnosis of, 235
influence of age in, 81	Gastritis, phlegmonous, 74
of alcohol, 86	Gastric ulcer, acute, 131
anæmia, 93	chronic, 183
Bright's disease, 91	Gastrostomy in cardiac stenosis, 290
cardiac discase, 91	Gastro-enterostomy, 283, 295, 309, 319
diabetes, 96	Geographical distribution of ulcer, 78
climate, 78	Glands, solitary, 116
excessive secretion, 88	Glands as cause of tumour, 212
food, 86	
heredity, 86	
hygiene, 85	Hæmatemesis, causes of, 159
lead poisoning, 96	diagnosis of, 160
malaria, 96	Hæmatemesis in acute ulcer, 132, 146
menstruation, 94	diet in, 170
occupation, 84	frequency of, 132
parturition, 96	symptoms of, 136
scurvy, 96	treatment of, 170
sex, 80	Hæmatemesis in chronic ulcer, 193
syphilis, 91	frequency of, 193
traumatism, 87	excessive, 199
tubercle, 90	moderate, 197
vascular disease, 91	rectal feeding in, 170
Evidences of former ulceration, 10	recurrent, 198
	slight, 195
	styptics in, 173
Fæcal vomiting, 356	surgical treatment of, 174
Fever, anæmic, 137	symptoms of, 195
in perigastric abscess, 325	tamponing in, 173
in peritonitis, 143	transfusion in, 174
Fistulæ, 68, 351	treatment of, 170
bigastric, 72	Hæmatemesis in duodenal ulcer, 213
duodenal, 72	frequency of, 213
gastro-duodenal, 72	Hæmorrhage from ulcer, 49
gastro-colic, 71, 365	frequency of, 50, 132, 193
gastro-cutaneous, 73, 363	mechanism of, 53
gastro-mediastinal, 70, 361	mortality from, 50
gastro-pericardial, 70, 357	source of, 50
gastro-thoracic, 68, 351	Hæmorrhage, venous, 51
Flatulence, treatment of, 251	Hæmorrhage, undetected, as cause of
Follicular ulcerations, 121	anemia, 380
Frequency of duodenal ulcer, 79	Hæmorrhagic form of ulcer, 221
of gastric ulcer, 78	diagnosis of, 237
Frost-bitc, 87	Healing, conditions which prevent, 125
Funnel-shaped ulcer, 19	Heart, perforation of, 52, 358
	symptoms of, 358
77	Heart disease in ulcer, 76, 90
Gall-Bladder, adhesions to, 31	Hepatic cirrhosis, ulcer in, 51, 154
perforation of, 67	Heredity in ulcer, 86
rupture of, 46	Horse-shoe ulcer, 14
Gastralgic variety of ulcer, 215	causation of, 16
diagnosis of, 230	Hour-glass stomach, 15, 44
Gastric contents in acute ulcer, 164	symptoms of, 291
chronic ulcer, 212	treatment of, 295
Gastrie diverticula, 46	Hyaline thrombi in vessels, 26
Gastric fistulæ, 68, 351	Hydrochloric acid in acute ulcer, 164
Gastric hyperesthesia, 162	chronic ulcer, 212
diagnosis of, 162, 231	Hygiene in ctiology, 85
Gastric paralysis, acute, 319	Hyperacidity in ulcer, 114

Hyperacidity, as cause of non-healing, 127	MALARIA as cause of ulcer, 96, 103, 128 Manganese, use of, 253
diagnosis of, 230	Mechanical causes of ulcer, 113
Hyperasthesia of stomach, 162	Mediastinum, perforation of, 70
symptoms of, 162	symptoms of, 361
diagnosis of, 162, 231	Melæna in duodenal ulcer, 157, 213
Hypersecretion, 299	frequeuey of, 213
eauses of, 299	symptoms of, 213
diet in, 308	in gastric uleer, 134, 193
digestion in, 306	symptoms of, 133, 193
diagnosis of, 307	treatment of, 170 as eause of anæmia, 380
gastritis frou, 303	Menstruation, disorders of, 94, 205
lavage in, 309	hæmatemesis with, 95, 194
prognosis of, 307	in relation to uleer, 94
signs of, 306 symptoms of, 301	Milk diet in acute ulcer, 167
treatment of, 308	in chronic ulcer, 243
urine in, 302	Milk, peptonised, 246
vomiting in, 301	Mortality in ulcer, 226
Hypodermic alimentation, 172	Multiple ulceration, 3
use of iron, 253	,
	Nyapogra gastuis sousse of 100
ILIAC abscess from ulcer, 337	Necrosis, gastric, causes of, 100
Infancy, gastric uleer in, 83	bacterial, 114 Neurasthenia in ulcer, 205
Inflammation as eause of ulcer, 108	
Inflammation of stomach after ulcer, 299	Neurotic eauses of ulcer, 113 Nervous dyspepsia, diagnosis of, 236
with ulcer, 74	Nervous disorders after ulcer, 311
thoracie organs, 62, 326, 351	Nervous symptoms with ulcer, 205
Intestinal obstruction after laparotomy,	Noma, gastric ulcer in, 150
180	Number of ulcers in duodenum, 3
Iodides, use of, 254	in stomach, 1
Iron, use of, 253	Nutrient enemata, 271
hypodermic use of, 253	composition of, 172
Irregular ulcers, 13	dangers of, 170
Kny orn of georg 26	
Keloid of scars, 36	OBLITERATION of bile duct, 46, 48, 295
	symptoms of, 295
LAPAROTOMY for hæmorrhage, 174	treatment of, 297
perforation, 175	Obstruction of gastrie arteries, 100, 103
indications for, 176	veins, 104
performance of, 177	Occupation, influence of, 84
results of, 180	Omental tumours, 209
sequelæ of, 179	Œsophagus, ulcer of, 42
Latency of duodenal ulcer, 212	symptoms of, 287
gastric ulcer, 220	stenosis of, 283
Lavage, dangers of, 194, 201	,
in læmatemesis, 173	
hypersecretion, 309	Pain in acute gastrie uleer, 138
pyloric stenosis, 282	duodenal ulecr, 212
Lientery, 366	character of, 213
Liver, adhesion of ulcer to, 29, 271	frequency of, 212
diagnosis of, 272	onset of, 213
symptoms of, 271	treatment of, 248
treatment of, 275	Pain in chronic gastric ulcer, 184
cirrhosis of, 76	access of, 184
as cause of hamorrhage, 154, 161	causes of, 190
erosion of, 167	character of, 184
hæmorrhage from, 50	effect of food on, 187
Localised peritonitis, simple, 143	effect of posture on, 188
suppurative, 58, 323	effect of pressure on, 187

Pain, radiations of, 186	Perigastric abscess, terminations of, 63,
situations of, 185	343
treatment of, 248	treatment of, 349
varieties of, 190	varieties of, 324
Pain in hypersecretion, 301	Perigastric adhesions, 28, 269
perigastrie abseess, 324, 327	effects of, 31, 269
perigastric adhesions, 271	pain from, 271
Panereas, adhesions to, 29	treatment of, 275
erosion of, 24, 66	vomiting from, 271
in vomit, 194	Peritonitis, acute general, 56
perforation of, 66	symptoms of, 142, 201
tumour from, 210	treatment of, 175
Pancreatic ducts, obliteration of, 46,	Pernieious anæmia, 380
48	Phantom tumours, 209
juice in stomach, 66	Phlegmonous gastritis, 74
Paralysis of stomach, acute, 319	Phthisis following ulcer, 370
Paradoxical dilatation, 295	symptoms of, 371
Parotid abscess, 334	varieties of, 371
Pathogenesis of uleer, 97	Physical signs of adhesions, 271, 272
Perforation of stomach, 55	eaneer, 379
eauses of, 55	stenosis of cardiae orifice, 287
diagnosis of, 163	dilatation of stomach, 279
frequency of, 55, 200 operation for, 175	gastrie fistulæ, 351
results of, 323	hour-glass stomach, 295
site of uleer in, 56	hypersecretion, 299
symptoms of, 139, 201	perforation of eolon, 365 diaphragm, 351
treatment of, 175	lung, 351
Perforation of eolon, 71, 365	pericardium, 357
duodenum, 72	pyloric stenosis, 279
diaphragm, 64, 68, 343, 351	Plastie perigastritis, 32
heart, 70, 358	symptoms of, 269
lung, 69, 351	Pleura, perforation of, by abscess, 64, 343
mediastinum, 70, 361	ulcer, 69, 351
perieardium, 70, 357	Pleurisy, secondary, 63, 351
pleura, 69, 351	Pneumonia, ulcer in, 149
skin, 73, 363	Pneumoperieardium, 70
viseera, 66	symptoms of, 357
Pericardium, perforation of, by abscess,	Pneumothorax from abseess, 64, 343
64	ulcer, 69, 352
symptoms of, 344	Pouches of duodenum, 47
Perieardium, perforation of, by ulcer, 70	stomach, 46
symptoms of, 357	Pressure necrosis, 107
Periduodenal abseess, 65, 323	Primary uleer of duodenum, 156
frequency of, 65	stomach, 131 Prognosis of acute duodenal uleer, 166
position of, 65	
results of, 66, 334 signs of, 341	gastrie uleer, 165 ehronie gastrie uleer, 225
treatment of, 349	hæmatemesis, 227
varieties of, 337	hypersecretion, 307
Perigastritis, simple, 143	perforation, 165, 228
plastie, 32, 269	tetany, 318
suppurative, 58	Pulmonary complications of absects, 62.
Perigastric abseess, 58, 323	343
boundaries of, 59	Pulmonary disease with uleer, 76
ehronie, 327	Pyæmia as eause of uleer, 101, 115
eomplications of, 62, 341	from uleer, 76, 341
diagnosis of, 345	Pylephlebitis, 343
frequency of, 58	Pylorie stenosis, 39
lateney of, 327	causes of, 39
signs of, 334	diagnosis of, 280
symptoms of, 324 , 327	frequency of, 39

. 11.1140

INDEX

Pyloric stenosis, symptoms of, 277
treatment of, 280
Pylorus, tumour of, 208
ulcer of, 11
Pyo-pneumothorax from absccss, 64,
343
subphrenicus, 323
ulcer, 70, 352
Pyrexia from hamorrhage, 137
perforation, 143

Rectal feeding, dangers of, 170 methods of, 172 Renal discase with ulcer, 76 Resorcin, uses of, 251 Results of cicatrisation, 38 symptoms of, 277 Retro-peritoneal abscess, 65 symptoms of, 337

Salt solution for transfusion, 174 in diagnosis, 207
Scars, frequency of, 33
appearances of, 10, 35
cancer of, 75
formation of, 37
keloid of, 36
ulceration of, 36
Sequelæ of ulcer, 269
Shape of ulcers, 13

Sex in duodenal ulcer, 81 gastric ulcer, 80 Silver salts, use of, 249

Situation of gastric ulcers, 5 duodenal, 12

perigastric abscess, 59 Size of ulcers, 3

Skin, perforation of, 73 Solitary glands, disease of, 116 Spirits in ctiology of ulcers, 86 Spleen, erosion of, 52, 67

Stenosis of bile duct, 295 Stenosis of cardiac orifice, 41, 283 causes of, 283

diagnosis of, 288 signs of, 287 symptoms of, 287 treatment of, 290

Stenosis of duodenum, 45 Stenosis of pylorus, 39

> causes of, 39 diagnosis of, 280 signs of, 279 symptoms of, 277

treatment of, 280
Stenosis of stomach, 44, 291
Stomach, acute paralysis of, 319

ach, acute paralysis of, 319 acute ulcer of, 131 blood supply of, 51 cancer of, 75, 378 Stomach, chronic ulcer of, 183 cirrhosis of, 74 contractions of, 30 deformities of, 38, 277 dilatation of, 38, 277 diverticulatof, 46 diseases of, in ulcer, 74 hour-glass, 44, 291 perforation of, 55, 139 scars in, 33 Styptics in hæmorrhage, 173 Subphrenic abscess, 58 symptoms of, 323 Surgical treatment of adhesions, 275 hæmorrhage, 174 hypersecretion, 309 hour-glass stomach, 295 perforation, 175 stenosis of cardia, 290 pylorus, 283 subphrenic abscess, 349 Syphilis in ulcer, 76, 91

Tamponing stomach, 173
Temperature after hæmorrhage, 137
perforation, 143
in perigastric abscess, 325
in ulcer, 205
over epigastrium, 207
Tetanus, 311, 315
Tetany of gastric origin, 311
etiology of, 316

treatment of, 254

Tetany of gastric origin, 311
etiology of, 316
frequency of, 311
mortality of, 315
prognosis of, 318
symptoms of, 311
treatment of, 319
varieties of, 312
Thermal causes of ulcer, 113

Thoracic complications of abscess, 63, 343

of ulcer, 69, 351 Thrombosis as cause of ulcer, 103 Transfusion after hæmorrhage, 174 Traumatic ulcer, 87, 113

symptoms of, 155 Treatment, climatic, 254 constitutional, 169, 2

constitutional, 169, 253 dietetic, 167, 243

local, 167, 248
Treatment of acidity, 251
acute ulcer, 167
adhesions, 275

anemia, 253 chronic ulcer, 243 constipation, 252 fistule, 356 flatulence, 251 gastritis, 309

hamorrhage, 170

hypersecretion, 308
loss of flesh, 254
pain, 248
perforation, 175
perigastric abscess, 349
pyloric stenosis, 280
stricture of cardia, 290
tetany, 319
vomiting, 251
Trophic theory of ulcer, 114
Turpentine in hæmorrhage, 173
Tubercle following ulcer, 370
varieties of, 371
Tubercle with ulcer, 76
Tumour with ulcer, 208

Ulcer, duodenal acute primary, 156 acute secondary, 158 appearances of, 13 chronic, 183 cicatrices of, 35 number of, 3 sequelæ of, 269 size of, 4 symptoms of, 156, 183 treatment of, 167, 243 Ulcer, gastric acute primary, 131 acute secondary, 146 chronic, 183 cachectic, 221 catarrhal, 218 dyspeptic, 220

hæmotrhagic, 221 latent, 220 morbid anatomy of, 1 pathogenesis of, 97 results of, 28 sequelæ of, 269 Urine in chronic ulcer, 206 hypersecretion, 302 Urine, suppression of, 206 Varices in stomach, 51 hæmorrhage from, 154 Vascular spasm, 106 Vena cava, erosion of, 53 Venous hæmorrhage, 51, 154 frequency of, 51 sources of, 51 Venous obstruction as cause of ulcer, Vomiting, causes of, 191 effects of, 193 in acute ulcer, 138 chronic ulcer, 191 duodenal ulcer, 213 gastritis, 303 hour-glass stomach, 292 hypersecretion, 301 paralysis of stomach, 320 pyloric stenosis, 277 treatment of, 251

Ulcer, etiology of, 78

gastralgic, 215

Vomiting variety of ulcer, 218 diagnosis of, 233

58

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Fenwick's (S.) Medical Diagnosis, 6

Obscure Diseases of the Abdomen, 9

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Treatise on Diseases of the Lungs 6

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Barnes (R.) on Obstetric Operations, 3
— on Diseases of Women, 3
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— Microscope in Medicine, 6
— Urinary and Renal Derangements, 12
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Bell on Sterility, 4 Bell on Sterility, 4
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Bentley's Systematic Botany, 5
Berkart's Bronchial Asthma, 6 Berkart's Bronchial Asthma, 6
Bernard on Stammering, 7
Bernays' Notes on Analytical Chemistry, 13
Bigg's Short Manual of Orthopædy, 9
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— Laboratory Teaching, 12
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Bowlby's Injuries and Diseases of Nerves, 9
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Brockbank on Gallstones, 8
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— Curvatures, &c., of the Spine, 9
— Talipes Equino-Varus, 9
— Dislocation of Hip, 9
Brown's (Haydn) Midwifery, 3
— (Campbell) Practical Chemistry, 13
Bryant's Practice of Surgery, 8
Bulkley on Skin Diseases 11
Burckhardt's (E.) and Fenwick's (E. H.) Atlas of Syphilis and the Nervous System, 7 Granville on Gout, 7 Green's Manual of Botany, 5 Greenish's Materia Medica, 4 Groves' and Thorp's Chemical Technology, 14 Guy's Hospital Reports, 7 Habershon's Diseases of the Abdomen, 7 Haig's Uric Acid, 6 — Diet and Food, 2 Harley on Diseases of the Liver, 7 Harris's (V. D.) Diseases of Chest, 6 Harrison's Urinary Organs, 11 Hartridge's Refraction of the Eye, 10 — Ophthalmoscope, 16 Burkhardt's (E.) and Fenwick's (E. H.) Atlas of Cystoscopy, 11
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Carpenter and Dallinger on the Microscope, 14
Carpenter's Human Physiology, 2
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Charteris' Practice of Medicine, 6
Chauveau's Comparative Anatomy, 14
Chevers' Diseases of India, 5
Churchill's Face and Foot Deformities, 9
Clarke's Eyestrain, 10 ——— Ophthalmoscope, 16

Hawthorne's Galenical Preparations of B.P., 5

Heath's Certain Diseases of the Jaws, 8

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[Continued on the next page.]

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Manual of the Diseases of Indi
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